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VISUAL FUNCTIONS IN STREPHOSYMBOLIA

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NEW YORK

Children who make little or no progress in learning to read during their first two or three years in school quite naturally are often referred to the ophthalmologist, and some of them undoubtedly need corrective lenses. However, many, if not the majority, of them have adequate vision and in reality present neurologic problems. One owes a considerable debt to two English ophthalmologists—Morgan, who described the first case of congenital word blindness in 1896, and Hinschelwood, who published a small monograph on this subject in 1917. In his report Hinschelwood included several cases of acquired word blindness, as well as a group of the congenital type, and emphasized the striking parallelism between them; because of this likeness, he ascribed the occurrence of the syndrome in children to a failure of development of the cerebral cortex in the neighborhood of the angular gyrus.

In 1926 I studied several cases of delay in learning to read and was struck by the inability of the children to differentiate "b" and "d" and "p" and "q" and their tendency to read many words from right to left, instead of in the usual direction, which led to uncertainty in their distinguishing such words as "was" and "saw" and "on" and "no." I found, also, that some of them showed an unexpected facility in reading mirrored print and that some also had a native skill in producing mirror writing. To account for these observations and to bring the whole syndrome into better consonance with advancing neurologic views concerning cerebral localization, I offered the theory that the condition in these cases was not the result of a pathologic factor, such as Hinschelwood's cortical agenesis, but a physiologic deviation due to failure of aquisition of the normal adult pattern of complete dominance of one hemisphere of the brain.

In cases of loss of acquired visual functions, three distinct steps, or levels, of elaboration in the brain can be recognized. The first permits only the seeing of objects of the environment but does not tell the meaning of the things seen; the second adds the meaning of objects, maps, pictures, etc., but it requires a third step—or relay—to permit the reading of words. These three steps are recognized by neurologists in the three clinical syndromes of cortical blindness, mind blindness and word blindness. It is generally believed that it is only at the third level that the principle of unilateral dominance applies, and there are reasons for believing that at this level the records, or engrams, in the two hemispheres of the brain are opposite in sign, or antitropic, to each other and that one engram must be elided to prevent confusion. This elision of the engram of the nondominant hemisphere seems to occur only at the third, or mnemonic level, since only here does a unilateral

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lesion completely destroy function, as can be seen in cases of acquired alexia. Learning to read, therefore, according to this envisagement, entails the elision of one of the two antitropic records, or engrams, and faulty or incomplete elision would result in uncertainty in mnemonic recall of orientation and progression, such as is seen in children with congenital word blindness.

It should be remembered that these reversals of form and direction are to be seen best during the first few years of learning to read, and that after continued exposure they largely disappear, although frequently in an older child the tendency to reverse can be demonstrated by using unfamiliar letter combinations, such as nonsense syllables. The confused memory patterns which interfere with recogni-

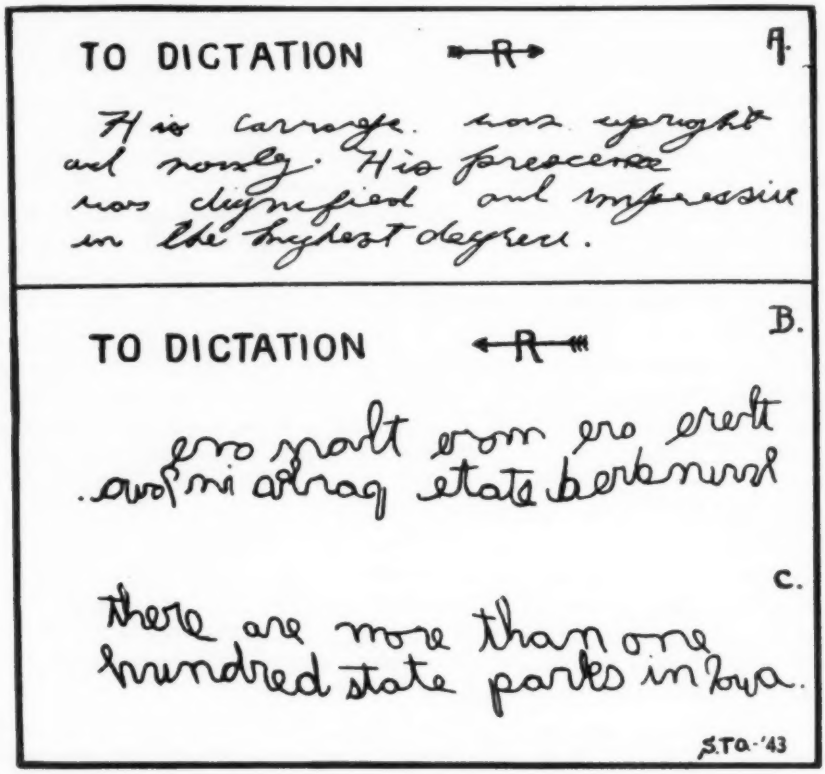
TO DICTATION
A.
His carriage was upright and manly. His presence was dignified and impressive in the highest degree.
TO DICTATION
B.
C.
there are more than one hundred state parks in Iowa.
S.T.O.-'43

Fig. 1.—*A*, tracing of the handwriting of an 11 year old girl, written to dictation with her right hand; *B*, a tracing of her mirror writing, also made with her right hand, after only two or three trials of this procedure, and *C*, a tracing of *B* reversed by printing through the back of a negative, so that the product can be more readily compared with *A*.

tion in reading are also to be seen in the same children when they attempt to spell, and reversals in spelling persist after the child has learned to read. The most striking evidence of antitropism comes from some persons with special writing disability, in whom skill in mirror writing lies dormant and unsuspected. One of my patients who could not learn to write legibly in the common, or dextrad, direction could produce creditable mirrored script with the first attempt at sinistrad progression.

This physiologic theory offers a much more favorable prognosis than did its predecessor, and the work of my associates and myself in retraining, as well as

extended observations during the last eighteen years, has, I believe, substantiated it. As descriptive of this syndrome in cases of reading and spelling disability I offered the term "strephosymbolia," or "twisted symbols," as being less misleading, especially to the layman, than the older term "congenital word blindness." To the neurologist the term "word blindness" means a loss or lack of the ability to recognize words although vision may be intact, but to the layman the term infers an inability to see. Moreover, I believe this disorder is hereditary rather than congenital, and I much prefer to call it developmental than either congenital or hereditary.

One of the visual functions which is often discussed in connection with reading is that of eyedness. There is a common misconception that the eye selected for sighting—the master eye—is the eye with the better vision. This is not always true. As an example, 1 of my patients wore a + 2.00 D. sphere and a + 1.00 D. cylinder for his right eye, while for his left eye a + 1.25 D. sphere and a + 0.50 D. cylinder was prescribed. In spite of the greater errors in his right eye, he used this eye consistently for sighting. The master eye is usually on the same side as the master hand, but cross preferences in either direction are to be found. Sighting is a function of the extrinsic ocular muscles, which act to bring the object observed, the pupil and the fovea centralis into line. These muscles are of the striated, or voluntary, type and are under the control of the motor centers of the brain adjacent to those which control handedness. It is possible that the right or left homonymous parts of the peripheral portion of the retina might have a superior equipment and so determine the eye most apt to be attracted to a distant object and thus fix the eyedness in early infancy before binocular fusion has begun. However, the frequency with which left eyedness and ambiocularity occurs in families in which there is also a preference for the left hand in some members leads me to the belief that eyedness is dependent on hereditary factors which govern the choice. It must be noted, however, that the many crossed patterns of eyedness and handedness would imply separate hereditary factors for the areas of the brain which control the eyes and those which control the arm.

The handedness pattern is open to much greater environmental influence than is eyedness, although there is evidence that the native eyedness pattern can be shifted also. Shooting a rifle was the factor in 1 such case, and my colleagues and I have observed children in whom a spontaneous shift from the left to the right eye has been noted in repeated examinations over a period of years. Some left-eyed persons can aim a rifle with the right eye only if the left eye is closed, or covered, while right-eyed persons usually learn to disregard the image in the left, or non-dominant, eye and can aim a rifle with both eyes open. In the case of a shotgun the situation is different, since without sights on the gun it may be aimed with the left eye. A priori, one might think that in aiming at a distance this would be a negligible factor, but that it does influence the accuracy of aim is recognized by gunsmiths, who build special stocks with a slight offset for those who shoot from the right shoulder but aim with the left eye. Accuracy in the use of a shotgun is said to be much improved by this procedure, and the same principle may apply in certain types of machine guns.

Some of the simpler tests of eyedness are not entirely trustworthy when used alone, and as a consequence my associates and I make use of a battery of tests to determine eyedness. In this connection it is interesting to note that the manoptoscope devised by Parsons, in our experience, frequently gives results out of harmony with those of other tests. Parsons used this instrument in his examinations on which he based his conclusion that left eyedness is evidence that a given child was originally intended to be left handed and, further, that eyedness determines which side of

the brain shall be the dominant one. He held that when left eyedness is found in a right-handed person it is evidence that training has influenced the handedness pattern and that the right-handed pattern is implanted in many naturally left-sided children because the right hand is used habitually by so large a proportion of both mothers and teachers. However, we have seen a number of left-handed children who are right eyed, whereas training right-handed children to use the left hand, either purposively or accidentally, is rare. Many persons, moreover, are neither definitively right eyed nor left eyed, and may be termed "ambiocular."

In 102 patients with reading disabilities whose records I have recently reviewed, the following distribution of eyedness patterns was found: right eyedness, 40 patients; left eyedness, 37 patients, and ambiocularity, 25 patients.

The left-eyed and the ambiocular persons together numbered 62, as against 40 who were right eyed—obviously a much higher proportion than one would expect in the population at large. Of the 40 patients who were right eyed, 12 were either left handed or had mixed right-handed and left-handed habits. Of the 37 who were left eyed, only 5 were left handed, but 17 showed a mixture of handedness patterns or gave a history of having been shifted to right handedness in childhood. Of the whole group of 102, 69 showed a crossed or mixed pattern of handedness and eyedness, and 83 gave a family history of left handedness or of language disorders.

My interpretation of these results is that left eyedness is not the cause of the reading disability but rather indicates that there is present in these persons a tendency toward use of the right hemisphere of the brain, i. e., left sidedness. This tendency may or may not involve those parts of the brain which are of primary importance in reading. The symptoms seen in the left-eyed and in the ambiocular patients do not differ from those seen in the right-eyed persons, and while covering the eye might lead to a change in eyedness, one would not expect any influence on the higher centers of the brain which are involved in strephosymbolia. Each macula is connected with both calcarine areas, and Sherrington's flicker experiments demonstrated that functionally the two eyes are interchangeable; it would seem therefore to make no difference which eye is used for sighting, since the image from either eye is probably relayed to the higher centers on both sides.

A second group of visual functions which is to be considered in connection with the reading disability includes the various refractive errors. There is no question that serious refractive disorders will interfere with reading, but many persons with minor degrees of myopia, hyperopia and astigmatism learn to read easily. Moreover, many persons with reading disability have normal refraction. When more serious errors are present, not only does their correction with appropriate lenses fail to enable the child with strephosymbolia to read without special instruction but even while he is wearing such lenses such a child exhibits the same errors of orientation and progression as do other children with strephosymbolia. This is, I believe, what one would expect on theoretic grounds. I have already mentioned the tendency to reversals in the mnemonic recall of previous exposures to the word, which must be used in order that it be recognized when it is presented again. This point is often difficult for the layman to grasp, since recognition of a word at sight seems to be simultaneous with seeing it. This, of course, is because the transmission of nerve impulses from the arrival platform to the region of the angular gyrus is too rapid to be registered in consciousness. The existence of a special zone of the cortex of primary importance in the function of word recognition is, however, clearly demonstrated in cases of acquired alexia, in which selective loss of the ability to read occurs without loss of the ability to see or of the capacity to

recognize objects, diagrams, maps, etc. These last functions are sometimes disturbed in cases of large occipital lesions which have involved the rest of the occipital cortex of both hemispheres, and acquired alexia is rarely, if ever, pure. A homonymous hemianopsia is frequent, but acquired hemianopsia can occur without alexia, and alexia is combined with it only when it involves the master hemisphere and when the lesion extends far enough forward to cut both the homolateral path from the calcarine cortex to the region of the angular gyrus and the heterolateral path from the opposite calcarine cortex by way of the corpus callosum (subcortical word blindness of Dejerine). This, again, demonstrates the adequacy of one hemisphere, since a lesion which cuts only the homolateral pathway does not cause alexia even though it is in the master hemisphere. While hemianopsia is the most common complicating picture, other symptoms, such as agraphia and varying degrees of aphasia, are also often found; indeed, in the acquired case pure alexia is so rare that some neurologists have questioned its occurrence. Persons with strephosymbolia, on the other hand, often show the alexia syndrome in exquisite purity. Handwriting may or may not be affected, and when it is not,

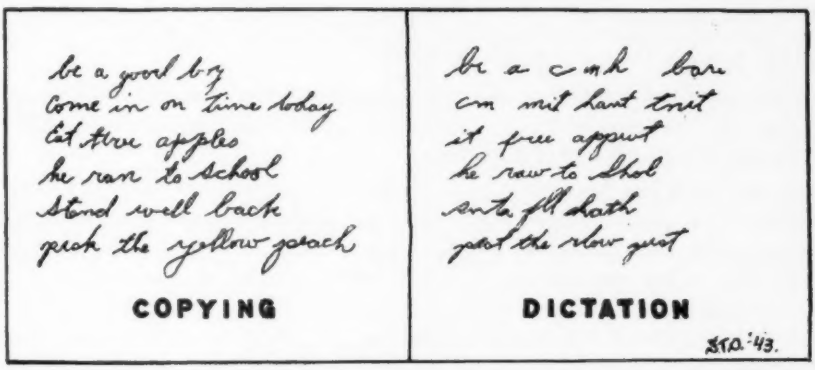

Fig. 2.—Carbon paper tracing of the writing of a 21 year old patient with strephosymbolia, showing the contrast between copying and writing to dictation (see "Addendum").

an illuminative demonstration of the mnemonic character of the disturbance and the adequacy of vision can be made by comparing the child's ability to copy accurately with the errors he makes in attempts to read or to write to dictation. In both these processes he calls on his visual memory, and in both he makes the typical errors of reversal. It is difficult to see how reading "was" for "saw," "ratshin" for "tarnish" or "astrep" for "repast," or how writing "ti" for "it" or "stpo" for "stop" could result from any refractive error.

The foregoing comments may, I think, be applied equally well to the heterophorias.

In a case in my experience, the diagnosis of hyperphoria was made at about the same time that I first saw the boy, when he was 11 years of age, but his family did not follow the ophthalmologist's advice and neither glasses nor treatment was provided. The boy had been in school most of the five preceding years, and while he did not have a very good basic equipment, he nevertheless had a mental age of 9 years when he was first seen and should have been able to read at the third or fourth grade level. He manifested, however, definite strephosymbolia, and his reading and spelling abilities were below the first grade norms. An intensive course of phonic retraining was instituted; after two and a half years of this treatment he had gained about a fifth grade skill in silent reading, and his oral reading and spelling were at about the fourth grade level. At this time he was seen by Dr. Conrad Berens, who found exophoria of 8 prism diopters and right hyperphoria of 1 prism diopter at 25 cm. The gain

in reading previously recorded had, however, taken place before treatment for his heterophoria was begun.

In another case, that of a boy seen recently in Philadelphia, a diagnosis of exophoria was made, and sight reading methods were recommended, with definite prohibition of the phonic training which has been found so serviceable in retraining persons with strephosymbolia. This boy's father and paternal grandfather were left handed, and his mother had been a poor student, although we were unable to determine whether or not this had been due to a reading disability. The boy himself definitely favored his right hand in almost all activities but was left footed and left eyed. He was 12 years old and had been in school the usual period of years. Psychometric examinations by a battery of tests gave mental age ratings varying all the way from 9 years 7 months to 13 years 2 months. Even at the lowest of these ratings, he should have been able to read at third or fourth grade levels, but his reading achievement was that of a very low first grade level. He copied a second grade paragraph without errors, but he misread "left" as "felt," "was" as "saw," "on" as "no," "nip" as "pen" and "ton" as "not." In attempting to spell to dictation a column of simple words, he wrote "ol" for "low," "sa" for "ask," "egt" for "get," "bolg" for "belong" and "awy" for "way." When he was asked to read a simple paragraph printed in mirrored type, he accomplished this almost as rapidly as he read the ordinary print, whereas a boy of his age who had gained normal reading skills would require at least ten times as long for the mirrored passage. At the time of these examinations he was wearing the glasses prescribed for him by the ophthalmologist, who advised the sight reading teaching and interdicted the phonic training. I believe that with continuance of that proceeding the boy's directional confusion will persist and that he can be taught to maintain consistent dextrad progression only by phonic training and dextrad analysis and synthesis of words from their phonic equivalents.

I am not personally familiar with the aniseikonic disturbances, but I understand from others that patients with such disorders are by no means all nonreaders, nor do the nonreaders all show that disorder.[1]

Much attention has been paid to vacillating movements of the eyes, and while bad habits in this regard might easily interfere with easy and fluent reading, it is also obvious that such a boy as the one last mentioned would scan a word from either direction in his confusion as to which way it should be read and would therefore exhibit back and forth movements of the eyes as the result of his uncertainty as to direction. Moreover, such vacillating movements would scarcely account for the confusion such children show between single letters, such as "b" and "d" and "p" and "q," or for the facility in reading mirrored print, in which constant sinistrad progress rather than alternating ocular movements is required.

Finally, the data we have assembled from the study of left handedness and of various language difficulties in the family stock of children who have a specific disability in learning to read and show the strephosymbolia syndrome give what to me is convincing evidence that such children represent intergrades between right-sided and left-sided familial tendencies and that the reading disability follows fairly definite hereditary trends. In this connection it is interesting to note that the reading disability and other language disorders are much more frequent in boys than in girls. In the current sample of 102 patients, 84 were boys and 18 girls. About the same distribution occurs for stuttering. In families with this disturbance there are also more than the expected number of left-handed members and persons with delayed speech; stuttering; reading, writing and spelling disabilities, and abnormal clumsiness (developmental apraxia). In the childhood histories of children who come to attention as presenting reading and spelling problems we not infrequently find indications of developmental deviations in their acquisition of speech and motor patterns which bear out the belief expressed herein that the strephosymbolia syndrome can best be explained on the basis of confused cerebral dominance rather than of abnormal vision.

1. Imus, H. A.; Rothney, J. W. M., and Bear, R. M.: An Evaluation of Visual Factors in Reading, Hanover, N. H., Dartmouth College Publications, 1938.

ADDENDUM.—After this paper was presented at the meeting of the academy, but before it was completed for publication, I studied the case of a man aged 21 with the strephosymbolia syndrome who, in spite of having "served time" through eight grades of the public schools in New York state, had acquired less than first grade reading skill. Figure 2 shows (1) his ability to copy from print into script, demonstrating that there was no agraphia, and (2) his floundering efforts to write from dictation. This contrast demonstrates clearly that the confusion lies in the mnemonic recall and not in visual distortions. This patient is now being given remedial instruction, and his eyes are being studied by Dr. Conrad Berens. A complete report of this case, together with the results of the remedial work and of Dr. Beren's study, will be published later.

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DISCUSSION

DR. CONRAD BERENS: I am sure that any one who has studied children with reading difficulties with or without motor anomalies and refractive errors must agree with Dr. Orton that the neuropsychologic aspects are most important and that cerebral dominance must be considered in all cases, whether or not motor anomalies and refractive errors can be demonstrated. In another paper by Dr. Orton, he said that strephosymbolia was a factor in 10 per cent of cases; it would be interesting to learn whether this is his opinion now. I have studied the history of 37 patients with reading disabilities and found 2 with a definite history of reversals. Four patients had crossed dominance. Four had convergence excess, 4 convergence insufficiency, 2 divergence insufficiency and 1 divergence excess. Hyperphoria, present in each, varied in degree. All the patients had third grade fusion. It was my impression that the motor anomalies and refractive errors of only 60 per cent were important.

There seems to be no question that the problem of conflict in cerebral dominance is important. At the same time, conflict in ocular dominance, for example a tendency to suppress the image of one eye or to suppress the images of the two eyes alternately in an attempt to maintain fusion, should also receive attention in this discussion. To my mind, it would be difficult to say in some cases whether the cerebral conflict produced by cerebral dominance is more important than the ocular conflict, which naturally has a cerebral conflict superimposed. It is not always easy to exclude certain motor anomalies, and there is no question that the ordinary tests of visual acuity and the usual tests for muscular imbalance may be insufficient without the Maddox rod test and the screen test with the eyes in the cardinal positions of gaze. In some cases the Marlow occlusion test seems to be necessary for diagnosis; it is necessary particularly in cases of corrected anisometropia in which there are associated reading difficulties. There is a certain amount of cerebral confusion when the patient attempts to fuse visual patterns of different size or quality, and it may be difficult for him to close one eye. I have found that other methods of excluding one eye in reading are often unsatisfactory.

Dr. Orton said that 1 patient selected the eye with the higher refractive error as the master eye, but he did not record the visual acuity, so that it is hard for me to determine whether this patient's vision was poor in the right eye. I think that it is generally conceded among ophthalmologists that patients usually select the eye with the better visual acuity as the master eye, although motor anomalies, refractive errors and hand dominance must play some part.

Dr. Orton's suggestion that hereditary factors are associated with eyedness may well be the truth in the light of what is known of hereditary ocular difficulties, especially visual and motor anomalies, and the tendency for these defects to be found in certain families.

As to Dr. Orton's observation that dominance may be shifted from one eye to the other, I have seen this in several cases in which not only squint but amblyopia and the scotoma of amblyopia have been transferred from one to the other eye.

There certainly is no question that ocular dominance plays a part in shooting. It is especially important in the aiming of a rifle or shotgun with both eyes open.

I agree with Dr. Orton that one should use several tests for the determination of eyedness. I have constructed a screen with a 1 cm. perforation to be held at arm's length with both hands and found it fairly accurate. The finger point test with the right and the left index finger is useful. MacDonald's idea of placing a bent rod at the end of a head band so that the arm and hand will not be brought into the picture when the sighting eye is being determined is worthy of further study. This test is possibly more accurate for determining the master eye. I studied the master eye in relation to the landing of airplanes in the last war, and my finding was similar to Dr. Orton's, that—if the tests used were valid—a certain number of persons are ambiocular.

Dr. Orton's interpretation of eyedness seems logical when he says: "Left-eyedness is not the cause of the reading disability but rather is to be looked on as indicating that there is a tendency toward use of the right hemisphere of the brain. This tendency may or may not involve those parts of the brain which are of primary importance in reading." According to his illuminating interpretation, covering one eye and forcing use of the other would not interfere with the higher brain centers in "strephosymbolia."

Dr. Orton's observation concerning refractive errors is of great interest. I am sure that many ophthalmologists have seen patients with high refractive errors who were able to read rapidly. One also sees patients who have no appreciable errors of refraction or motor anomalies demonstrable by the tests usually employed who are handicapped in their ability to read. Many of the poor readers I have seen who have had fusion disabilities and were referred for examination for this reason either had only minor errors of refraction or a low degree of heterophoria. In my experience, anomalies of accommodation, especially anisoaccommodation and rapid fatigue of accommodation, have interfered more with the function of reading than low degrees of heterophoria and ametropia. The patient (13 years of age) with hyperphoria referred to me by Dr. Orton whose reading ability was improved by his phonetic training had 1.75 prism diopters of right hyperphoria with the Maddox rod and 3.5 prism diopters of right hyperphoria and 4.5 prism diopters of exophoria after the Marlow occlusion test. The hyperphoria was less with the eyes in the reading position, and only 1.5 prism diopters of right hyperphoria at 25 cm. was measured after the Marlow occlusion test. The accommodation was remote for the patient's age, 150 to 300 mm., and it fatigued in ten minutes with the right eye and in six minutes with the left eye with several rests. The near point of convergence was 90 mm. at the first examination. After two months of fusion training this was 45 mm. and the prism convergence had improved from 12 to 20 prism diopters.

What bearing the heterophoria and dysaccommodation may have had on the reading disability is difficult to evaluate without a knowledge of the original findings and without a reading test of each eye separately and of the two eyes together. One should also know what the ocular movements showed while the patient was reading and whether the hyperphoria became more evident when he became fatigued, as it has been shown to do in certain patients subjected to anoxemia tests. The bearing of the allergic condition and the general physical condition is hard to evaluate from the data presented.

Dr. Orton's concept of difficulties of orientation as caused by strephosymbolia seems to be logical and is the best explanation for reversal in mnemonic recall of previous exposures to a word that I know of, and his reference to the special zone of the cortex affected in acquired alexia has certainly been amply demonstrated pathologically. The fact that this difficulty is confined to reading, without

affecting the recognition of objects, is apparently more than suggestive. However, I know of no pathologic studies in congenital alexia, and I should like to know whether Dr. Orton is aware of any.

He certainly clarified the findings in hemianopsia for me, and the fact that the picture is usually complicated if the strephosymbolic patient shows pure alexia is most illuminating.

That writing may or may not be affected is a point in favor of his theory. I agree that the reversal of words in reading and writing is not a function of visual acuity and motor anomalies alone and must call on some other theory for explanation; Dr. Orton's is the most satisfying I know of.

My observation in cases similar to that of the Philadelphia boy agrees with Dr. Orton's, because psychologic tests usually show a high intelligence quotient and some of the worst readers have a mental age well above their chronologic age.

Dr. Orton brings up the question of vacillating movements of the eyes and points out that some of these unusual associated movements may be caused by the child's not knowing which way to read a word rather than by any motor incoordination. I believe this may be true in a limited number of cases, but to diagnose the presence of these unusual movements and determine their cause is often a baffling problem which requires the most careful studies. Tests under low oxygen tension with photography of the ocular movements may be required for a diagnosis of some of these movements (McFarland, R. A.; Knehr, C. A., and Berens, C.: The Effects of Anoxemia on Ocular Movements While Reading, *Am. J. Ophth.* 20: 1204-1221, 1937).

Dr. Orton has provided much food for thought, and, as is usual when any subject is discussed from the standpoint of another specialty, much has been learned. However, no acquisition of knowledge should be too one sided, and I hope that Dr. Orton's listeners will study their patients from the standpoint of strephosymbolia but also have a complete diagnosis of their ocular, medical, neurologic and psychologic problems, with special emphasis on motivation, not overlooking hereditary factors.

Dr. George H. Hyslop (by invitation): I did not have the advantage of reading Dr. Orton's manuscript, and I missed the first portion of his talk. I heard enough, however, I think, to make two or three remarks which are perhaps supplementary to what Dr. Berens has said. The points I should like to stress are these:

In attempting to judge a patient's performance in the use of either motor or sensory functions as the application of his intelligence to them, one must consider not only the primary pathway apparatus but the higher cerebral integration. Secondly, I think Dr. Orton is the originator of the concept (though he may not entirely agree with me) that certain types of impaired language function involve not a lesion or a structural defect but an inherited functional defect. This inherited functional defect may be imitated by an acquired lesion which does affect the inborn ability of the two hemispheres to coordinate properly.

The third point is that there is no necessary correlation between the results of ophthalmologic examination and the way the patient uses his eyes, not only in reading but in judging any objects which are in the field of vision.

The last point (I do not know whether Dr. Orton would have given more attention to it if he had had more time) is that in examining children who are presumed to have defective vision because they do not read, one may note disorders of integration through studying how they use their eyes and understand the meaning of objects which are similar in size but do not involve the language function. Thus one may get a clue that there is a disorder of integration rather than of vision. A disorder of refraction or primary perception will not manifest itself as a failure of conception of a limited or specific type of object.

Dr. Bernard Fread: It has been a privilege to hear so distinguished an authority on reading difficulties as Dr. Orton, whose name is well known not only

to ophthalmologists but to educators and other persons interested in this field. I was particularly interested to hear him mention that eyedness is not so much an inheritance difficulty as the educators have indicated, and I should like to ask him whether he feels that mixed dominance, that is dominance of the left eye and right-handedness, is a factor in reading difficulties.

The main thing Dr. Orton stressed, which seems important, especially to ophthalmologists, is that one must have the proper physical equipment to read; that is, one must have one's optical error and muscle imbalance corrected adequately to have the proper anatomic and physiologic basis for good reading. Dr. Orton mentioned the nature of hemianopsia and its effect on reading. There may be reading difficulties because of generally contracted visual fields. Also, one has to remember there is rarely one cause for difficulty in reading and that a great deal of investigation is needed, of which some is done by the ophthalmologist and a great deal by the properly trained psychologist and educator. In other words, the causes of any one patient's difficulty in reading must be multiple. One must not forget the fact that poor hearing and muscular coordination influence reading, which may also be difficult for children with defects of speech. The factors of illness, malnutrition, paralysis and disorders of the endocrine glands may influence reading by retarding the child's physical and mental development. I must not forget the value of general intelligence and mental development, because those factors also influence reading. An interesting point sometimes forgotten is that mirror reading and mirror writing, some educators feel, comprise an actual physiologic stage in the learning process of reading and need attention only when they persist.

In conclusion, I am grateful to the essayist and to Dr. Berens for emphasizing the fact that ophthalmologists should make careful studies of the accommodation mechanism and then refer their results to an educator interested in reading difficulties.

DR. LAURETTA BENDER (by invitation): May I ask what is the status of brain wave studies in regard to cortical dominance?

DR. SAMUEL T. ORTON: I do not think I have much to offer in closing the discussion. Dr. Berens spoke of finding reversals in a small number of the nonreaders he studied. I would assume that he saw older children or adults, and I would expect that. As one attempts to teach a child, the child gradually gets the direction straightened out, but still there is enough confusion in direction so that the recall of a word is not accurate. One finds that the more accurate recall frequently lags behind the amount of recall necessary for recognition of a word, so that the child learns to read before he learns to spell. Persons with reading difficulties are usually poor spellers.

Dr. Berens spoke of the case to which I referred in my paper. I have no record of the visual acuity. I gave all the details I had.

He also spoke of testing for eyedness with a screen with a hole in it. I have been using a comparable test for quite a while. A card about 6 by 8 inches (15 by 20 cm.) with a hole in the center somewhat smaller than a dime is used. It is offered first to the child with the examiner holding it with the hole over the bridge of the nose, so that the child must shift the head to bring one eye into line. Then the child is asked to hold it, first with one hand, then with the other and then with both. Occasionally one finds that the hand used influences the choice of the sighting eye, but commonly if the child has a dominant eye he will shift the card over in front of that eye. One finds some unusual responses, but I feel that with first the examiner and then the child holding the card one generally gets valuable data to be added to the results of the other tests used for eyedness.

Dr. Berens asked about the pathologic observations. There are none yet. I have done a great deal of work in brain pathology and have examined 1,500 or more human brains, and among these I have not yet seen a selective agenesis of the

type that Hinschelwood suggests. I have seen one hemorrhage in the angular gyrus in an infant's brain in which there were multiple hemorrhages.

In a survey of a whole community my associates and I found reading disabilities sufficient to retard development at least one year in 10 per cent of the total school population. If this proportion was true and reading difficulties were due to agenesis one certainly would find such a defect in the angular gyrus frequently in a series of 1,500 brains. Such a fault in development in the region of the angular gyrus would have to be present as a bilateral lesion in children to cause the reading disability, and I have not observed a brain with this selective agenesis in even one hemisphere.

Dr. Hyslop spoke of the matter of integration, and that is a point of great importance. I mentioned the fact that it is only when integration takes place at the third, or word, level that reversed memory pictures come out, and it is in the integration process that these reversals occur.

Dr. Fread asked a question concerning mixed dominance. I thought I had expressed the opinion that the mixture of dominance is not a causal factor but does give evidence that the child is not all right sided or left sided as far as the brain is concerned, and I am coming to the conclusion that there are a lot of persons who are not entirely one sided, who do not have all the motor functions organized in one hemisphere, in whom the dominance for some functions is in one hemisphere and that for some in the opposite. I am supported in this belief by observations made by Dr. Nielsen, a neurologist of Los Angeles.

Dr. Fread also brought up the question of intelligence. Of the 102 persons on whom I reported, none had an intelligence quotient below 90. All patients with an intelligence quotient below 90 were ruled out so that confusion due to the problem of intelligence would be excluded.

I agree with Dr. Fread as to the fact that reversals are present in almost all children when they are first learning to read. Almost all children are confused at first between "b" and "d," etc., but the confusion does not persist as it does in children with strephosymbolia.

In answer to Dr. Bender's question, I may say that I have not had an opportunity to work in the field of electroencephalography as yet but hope to soon.

TRAUMATIC ENOPHTHALMOS

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Although fracture has always been regarded as a cause of enophthalmos, there exists in the literature considerable speculation on the mechanism of the displacement of the eye when there is no external evidence of fracture. Most of the discussion on the sometimes distressing consequences of a blow on the eyeball took place before the roentgen rays were developed to a high point of usefulness. To many of the early observers fracture meant fragmentation and gross deformity. It was inconceivable to them that the eyeball could receive, withstand and transmit a blow forceful enough to fracture any of the walls of the orbit. Since means of demonstrating deep, or internal, fractures were lacking and, in later years, since roentgenography was not employed at its greatest efficiency, enophthalmos in many cases was considered an extraordinary phenomenon. Today, with the roentgen rays, one is able to study the orbit in fairly minute detail, and considerable knowledge of the changes which occur in this cavity in disease and in injury has been gained. With roentgenography the cause of enophthalmos has been clarified, and another indication of the value of the roentgen rays in ophthalmic practice has been added to a growing list.

In a recent reference to traumatic enophthalmos, Benedict [1] listed the following causes as responsible for varying degrees of enophthalmos:

1. Dislocation of the trochlea.[2]

2. Atrophy of orbital fat (trophoneurotic theory of Praun [3] and Beer,[4] mentioned by La Grange,[5] Gessner,[6] Lukens [7] and others).

3. Enlargement of the orbit (Lang [8] and others).

4. Cicatricial contraction of retrobulbar tissue (Praun,[3] La Grange,[5] Lederer,[9] Le Roux [10] and Luniewski [11]).

5. Rupture of orbital ligaments or fascial bands (Kilburn,[12] Fick,[13] Collins,[14] Shoemaker,[15] Morax [16] and Rand and Reeves [17]).

Read before the American Ophthalmological Society, Hot Springs, Va., June 11, 1943.

From the Institute of Ophthalmology of the Presbyterian Hospital, and the Department of Ophthalmology, Columbia University College of Physicians and Surgeons.

1. Benedict, W. L., in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 330.
2. Hughes, W. L.: Personal communication to the author. In the operation of recession of the trochlear nerve introduced by Hughes, enophthalmos was not of measurable degree in any of the cases.
3. Praum, E.: Die Verletzungen des Auges, Wiesbaden, J. F. Bergmann, 1899.
4. Beer, T.: Arch. f. Augenh. **25**:315, 1892.
5. La Grange, F.: Les fractures de l'orbite, Paris, Masson & Cie, 1917.
6. Gessner, C.: Arch. f. Augenh. **28**:297, 1888.
7. Lukens, C.: Ophthalmology **3**:30, 1906.
8. Lang, W.: Tr. Ophth. Soc. U. Kingdom **9**:41, 1889.
9. Lederer, R.: Arch. f. Augenh. **53**:241, 1902.
10. Le Roux, H., and Maklakoff: Arch. d'opht. **24**:176, 1904.
11. Luniewski, S. B.: Post. okul. **5**:47, 1903.
12. Kilburn, H. W.: Arch. Ophth. **31**:384, 1902.

6. Fracture of orbital wall (Lederer,[9] Lang,[8] Praun [8] and others).

7. Displacement of part of orbital tissue (Lang [8] and others).

As enlargement of the orbit and displacement of part of the orbital tissue could occur only as a result of fracture, three of the aforementioned points pertain to deformity of the bony orbit resulting from violence. Lang's contention that fracture occurred in every case of enophthalmos and that the displacement was probably due to all three of these conditions corresponds precisely with my experience. Lang, in discussing his 2 cases in 1889, stated:

> I would suggest that the injury may have produced a fracture and depression of a portion of the orbital wall; the orbital fat would then be no longer sufficient in quantity to fill this enlarged postocular area without a sinking in of the globe from atmospheric pressure and the resulting limitation in the ocular movements. These cases might then be considered as less exaggerated but similar to those of von Becker and Tweedy.

In von Becker's and in Tweedy's case the injury was produced by a cow horn's driving the globe into the antrum through a perforation of the floor of the orbit. In both cases the eyes were lost from view. Lang's opinion was stated without aid of surgical exploration, autopsy examination or, of course, roentgenography.

MATERIAL

Of 120 cases of fracture of the bones of the face in which the orbit was involved in some way, enophthalmos was present, or developed subsequent to the injury, in 53 (table 1). Fracture of the orbit was noted in every case of traumatic enophthalmos through a period of ten years. In 29 cases external deformity or severe fracture involving the margin of the orbit, with dislocation of fragments, occurred and the cases are not included in this study. In these cases fracture was obvious clinically and/or roentgenographically. In 24 cases the fracture was internal, or deep in the orbit, unassociated with solution of continuity or deformity of the margin of the orbit or of surface structures. This series of 24 cases (table 2), of the type which was inexplicable to the older authors, forms the basis of this report.

In all these 24 cases there was displacement of the eye of 1 to 9 mm. without other external deformity, as measured by the Hertle exophthalmometer. In most of the cases the eye was displaced posteriorly 2, 3 or 4 mm. In the cases of more severe injury, of the 24, the eye was displaced posteriorly and inferiorly. In 2 instances the eye was displaced posteriorly and slightly nasally. The displacement of the eye would have been overlooked clinically in a number of cases if the exophthalmometer had not been used on the suggestion of the roentgenographic report after the signs of recent trauma had subsided.

In the cases of severe displacement enophthalmos was apparent early. In 1 case in which I made examination the day after injury, the eye could scarcely be seen when the greatly swollen lids were pried open. In 2 other cases in which the enophthalmos was not severe, the displacement was apparent on the third and fourth days, when examination was made after the roentgenographic report was rendered. But, in most of the cases, because of hemorrhage, edema and/or air in the orbit, the displacement was not found until two or three weeks had passed.

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13. Fick, A. E.: Diseases of the Eye and Ophthalmoscopy, Philadelphia, P. Blakiston's Son & Co., 1896.
14. Collins, E. T.: Brit. M. J. **2**:846, 1899.
15. Shoemaker, W. T.: Am. Ophth. **9**:391, 1900.
16. Morax, M. V.: Bull. et mém. Soc. franç. d'opht. **47**:391, 1934.
17. Rand, C. W., and Reeves, D. L.: Surg., Gynec. & Obst. **69**:460, 1939.

The eye was exophthalmic at first in several cases. In general, it may be said that the severity of the early signs of violence to the eye bears no relation to the degree of injury delivered to the walls of the orbit.

TABLE 1.—*Data on Cases of Fracture of the Orbit Studied Roentgenographically Through a Ten Year Period*

Total number of cases of fracture		120
Total number of cases of traumatic enophthalmos		53 (44%)
Cases of combined external and internal fracture	29	
Cases of internal fracture	24 (45%)	

TABLE 2.—*Essential Data on the Twenty-Four Cases of Internal Fracture of the Orbit Causing Enophthalmos* *

Case No.	Patient	Injurious Agent	Degree of Enophthalmos, Mm.	Walls Fractured	Depression of Floor into Antrum, Mm.	Eye † Involved and Visual Acuity	Diplopia Present	Permanent Injury to Eyeball
1	F. B.	Fist	3	Inferior nasal	9	O. S. 20/20	Yes	None
2	E. W.	Fist	2	Inferior	6	O. D. 20/30	No	None
3	A. W.	Fist	9	Inferior	Complete	O. D. 20/30	Yes	None
4	L. T.	Fist	5	Inferior nasal	15	O. S. 20/20	Yes	None
5	L. A.	Fist	3	Inferior nasal	8	O. S. 20/20	No	None
6	O. J.	Fists	O. D. 4 O. S. 6	Inferior Inferior	14 16	O. D. 20/15 O. S. 20/15	Yes	None
7	J. C.	Ski	5	Inferior nasal	14	O. S. 20/50	Yes	Hole in fovea; partial optic nerve atrophy
8	J. B.	Automobile accident	6	Inferior	18	O. S. 20/20	Yes	None
9	J. K.	Fist	3	Inferior nasal	8	O. S. 20/200	No	Retinitis sclopetaria
10	C. S.	Gouging	1	Inferior	4	O. S. 20/40	No	Hemorrhage in vitreous; chorioretinal atrophy
11	G. T.	Knee	2.5	Inferior	7	O. D. 20/20	No	None
12	H. M.	Fist	2	Inferior nasal	7	O. S. 20/30	No	None
13	E. H.	Cow's horn	7	Inferior nasal	20	O. S. 20/20	Yes	None
14	S.	Fist	3	Inferior	10	O. D. 20/20	No	None
15	W. W.	Automobile accident	2	Inferior nasal	7	O. S. 20/20	No	None
16	W. R.	Fist	4	Inferior nasal	10	O. D. 20/40	No	None
17	M. C.	Fist	6	Inferior	14	O. D. 20/30	Yes	None
18	J. L.	Baseball	2	Inferior nasal	5	O. S. 20/30	No	None
19	S. C.	Automobile accident	3	Inferior	8	O. S. 20/20	No	None
20	W. W.	Fist	7	Inferior	Complete	O. S. 20/70	Yes	Paralysis of third nerve; partial optic nerve atrophy
21	A. W.	Golf ball	3	Inferior	6	O. D. 10/200	No	Retinitis sclopetaria
22	M. G.	Fists	3.5	Inferior nasal	9	O. S. 20/20	Yes	None
23	E. M.	Automobile accident	2	Inferior	6	O. S. 20/20	Yes	None
24	T. T.	Fist	4	Inferior	12	O. S. 20/20	No	None

* In 14 instances the fracture was due to the blow of a closed fist. The floor was fractured in all cases, and the nasal wall in addition in 11 cases. On calculation it was found that 1 mm. of enophthalmos indicated an average depression of 3 mm. of the floor of the orbit into the antrum. Diplopia was experienced in 11 cases. Permanent impairment of vision of the eye of the involved orbit was suffered in only 5 cases.

CAUSES OF FRACTURE

The circumstances responsible in these cases of internal fracture of the orbit were varied. It may be a matter for some surprise that in 14 instances the fracture was produced by a blow of the fist. Many of the patients were treated first for the ordinary "black eye." In most of the cases in which roentgenographic studies were made for this complaint fractures were found, and in those which

could be followed enophthalmos of 2 to 9 mm. developed. In 1 of the cases of most severe enophthalmos (case 3) the displacement resulted from the blow of a fist, and the case will be described in detail. In case 2 fracture of the floor of both orbits with bilateral enophthalmos resulted from an assault. Many of the patients with this injury were uncooperative, so that final notes were difficult to obtain; otherwise, this series would have been many times larger. Automobile accidents were responsible in 4 of the cases. In 3 cases the injury was received while the victim was playing football. In 1 case the patient was struck by a baseball; in 1 by a ski; in 1 by a golf ball, and in 1, by a cow's horn.

ROENTGENOGRAPHIC FINDINGS

The roentgenographic findings were characteristic. In every case the floor of the orbit was broken through into the maxillary sinus below. Fragments could usually be seen in this sinus, and the increased density of orbital tissue was always demonstrable, the tissue prolapsing in some degree into it. In the cases of severe enophthalmos the antrum was densely and completely clouded by the orbital tissue which filled it. The thin fragments of bone displaced into the antrum in these cases were the only trace of the floor of the orbit that could be found. In several of the cases of less severe displacement the infraorbital canal seemed to have braced the floor and stopped the descent of tissue. The degree of prolapse and the resulting enophthalmos seemed in several cases to have been determined in part by the size of the antrum. In the cases of severe displacement the antrums were large. In 15 of the cases deformity of the nasal wall of the orbit was apparent, in addition to fracture of the floor. The ethmoid cells were compressed by the medial displacement of the lamina papyracea in these cases. In all the cases the effect of the fractured walls was to increase the capacity of the orbit.

Fracture or deformity of the floor of the orbit can be recognized in the roentgenogram of the skull in the Caldwell position, especially when corresponding parts are compared. The degree of prolapse into the antrum can be observed better in the Water position. Good roentgenographic technic is essential.[18] The clouded appearance of the antrum caused by the presence of orbital tissue may be mistaken for the appearance most commonly produced by sinusitis. Obviously, fragments of bone are never seen in the antrum involved by the latter condition.

MECHANISM

The mechanism of the internal orbital fracture responsible for the posterior displacement of the eye is quite clear. From the nature of the fractures it is evident that the force of the blow received by the eyeball was transmitted by it to the walls of the orbit, with fracture of the more delicate portions. That the floor is always involved is quite understandable. The posterior convex portion of the floor bulges upward back of the eyeball in a position to receive most of the force transmitted by the eye. The floor is of very thin bone, similar in weight to the lamina papyracea —a fact easily demonstrated by transillumination of the dried skull—and is braced but slightly by the infraorbital groove, or canal. In the cases of less severe enophthalmos the posterior portion is fractured, and in the cases of more severe displacement the entire floor is broken through. It is usually in the latter cases that fracture of the lamina papyracea is also observed. The effect of these fractures is to increase the capacity of the orbit, and through the fracture of the floor tissue is

18. Pfeiffer, R. L.: Tr. Am. Ophth. Soc. **39**:492, 1941

allowed to prolapse into the antrum, with resulting enophthalmos. If the antrum is large enough, it is possible for the eye to drop into it and disappear from view.

In all of the 24 cases the force of the blow was concentrated in a small area, so that it struck the eye directly and was not buffered by the protective projecting structures of, or the structures adjacent to, the margins of the orbit.

SYMPTOMS

In half the cases, except for the acute signs of injury, distressing symptoms other than the displacement of the eye were not experienced. In 11 cases the patient did not know that the eye had been displaced, and in these cases there were no other symptoms. Limitation of motility occurred, and diplopia was bothersome, in 11 cases. In 5 cases paresthesia of the face resulted from the fracture of the infraorbital canal and rupture of the infraorbital nerve. This symptom soon seemed to disappear from the patient's mind.

That the eye is able to withstand such severe trauma and retain its visual function, as was true in most of the cases, is indeed remarkable. Central vision was destroyed in only 4 cases. In 1 case there was a hole in the macula; in 1 case, a hemorrhage in the vitreous, with unknown termination, and in the remaining 2 cases, rupture of the choroid and disorganization of the central area of the retina. In several instances reduction of visual acuity occurred to some degree. Dislocation of the crystalline lens was not noted, and detachment of the retina did not occur during the follow-up period. In many cases, however, the retina showed evidence of contusion when it was first examined. Commotio retinae, retinal and preretinal hemorrhages, iritis, dilatation of the pupil, subconjunctival hemorrhage, hypotony, edema and ecchymosis of the eyelids and orbital emphysema were the most common early signs. Rupture of the eyeball was not noted.

ILLUSTRATIVE CASES

For purposes of illustration, several cases are described briefly, with reproduction of the roentgenograms. The first case is fairly characteristic of most of the series; the second was one of bilateral enophthalmos, and the third was an instance of severe deformity.

CASE 1.—F. B., a woman aged 30, had been struck on the left eye with a closed fist in an altercation with her husband four months before. The eye was greatly swollen and was black and blue for days. Since this episode the eye had seemed to the patient to be "in too far." There was no double vision. Numbness of the left side of the face was no longer bothersome (fig. 1).

Examination.—The left eye was set deep in the socket, with a hollow space beneath the eyebrow. The pupils were equal and active on the two sides.

Vision was 20/20 — 3 in the right eye and 20/20 — 1 in the left eye.

The exophthalmometric reading was 18 mm. for the right eye and 15 mm. for the left eye. The palpebral fissure measured 9 mm. in width in the right eye and 7 mm. in width in the left eye.

There was orthophoria for near vision. No diplopia was brought out with the red glass. There was good motility in each eye.

The fundi were normal.

Roentgenographic Report (fig. 2).—Stereoscopic roentgenograms of the orbits showed that the margins were symmetric and of the mesoseme type. The sphenoid ridges, the superior orbital fissures and the temporal lines were also symmetric. The floor of the left orbit was fractured and was displaced inferiorly at least 1 cm. into the antrum. The nasal wall of the left orbit was concave and displaced nasally, compressing the ethmoid cells. The capacity of the left orbit, therefore, was much increased. There was no external evidence of fracture of the bones of the face. The right orbit was normal. The optic canals were circular, measured 5 mm. in

diameter and were symmetric. All the paranasal sinuses were clear except the left antrum, which showed prolapsed orbital tissues.

Impression.—The impression was that of fracture of the left orbit, involving the floor and the nasal wall, with prolapse of orbital structures into the left antrum, this displacement accounting for the enophthalmos.

Diagnosis.—The diagnosis was enophthalmos of the left eye, resulting from internal fracture of the orbit.

Advice.—The patient was not bothered sufficiently by the displacement to feel that an operation to restore the eye to a normal position was justified.

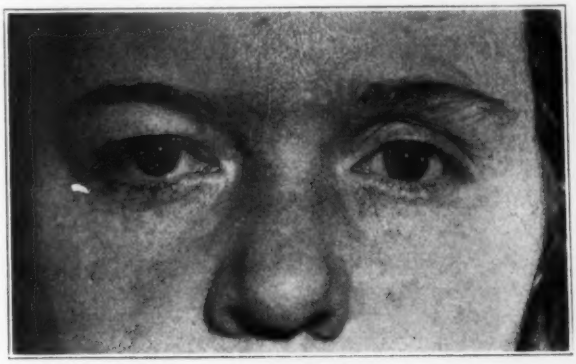

Fig. 1 (case 1).—Photograph of patient, showing the enophthalmos of 3 mm. of the left eye.

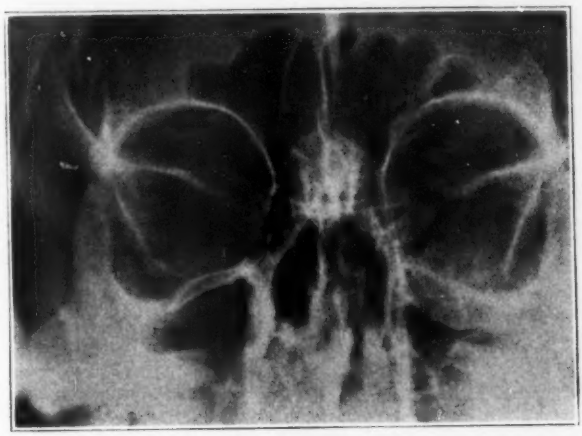

Fig. 2 (case 1).—Roentgenogram of orbits, showing the fracture of the floor of the left orbit, with prolapse of fragments and of orbital structures 9 mm. into the antrum.

CASE 2.—O. J., a Negress aged 23, had been attacked three years before, when she suffered severe blows to the face and eyes. Since the attack the eyes had seemed sunken, and double vision had been persistent, especially on looking upward.

There was weakness of the left superior rectus muscle, with pronounced secondary overaction of the right inferior oblique muscle.

Examination.—Vision was 20/20 in the right eye and 20/30 in the left eye; with correction it was 20/15 in the right eye and 20/15 in the left eye.

The exophthalmometric reading was 14 mm. for the right eye and 12 mm. for the left eye.

The interpalpebral fissure measured 6 mm. in width in the right eye and 5 mm. in width in the left eye.

Double vision was present and increased when the eyes were turned up and to the left. The fundi were normal.

Roentgenographic Report (fig. 3).—Roentgenograms of the orbit showed the margins to be symmetric, uninterrupted and of the mesoseme type. The sphenoid ridges, the superior orbital fissures and the temporal lines were also symmetric. The floors of both orbits were fractured, and fragments of bone could be identified in the antrums below, with prolapse of orbital tissue, approximately 18 mm. or more, into each antrum. The lamina papyracea of the left orbit was deformed with compression of the ethmoid cells posteriorly. The optic canals were nearly circular, measured 5 mm. in diameter and were symmetric and normal.

Fig. 3 (case 2).—Roentgenogram showing fracture of the floors of both orbits, with prolapse of fragments and orbital structures into both antrums.

Fig. 4 (case 3).—Photograph of patient, showing severe enophthalmos of the right eye, resulting from the blow of a fist.

Diagnosis.—The diagnosis was bilateral traumatic enophthalmos, more severe on the left side.

Operation.—Myectomy of the right inferior oblique muscle was performed.

Result.—The diplopia was relieved and the patient was content with the result.

CASE 3 (fig. 4).—A. W., a woman aged 25, was struck on the right eye by a fist in February 1942 and was unconscious for a few minutes, with some bleeding from the nose and mouth. Numbness on the right side of the face resulted from the blow; at the time of examination it was less bothersome. The eye was swollen shut for three weeks; when the swelling subsided, the sunken condition of the right eye was first observed. This progressed until July 1942. Vision had been preserved, but double vision was most annoying.

Examination.—The right eye was displaced posteriorly and inferiorly. A deep hollow space lay beneath the brow. The pupils were active and equal on the two sides.

Vision with correction was 20/30 + 2 in the right eye and 20/30 in the left eye. Exophthalmometric readings were 9 mm. in the right eye and 18 mm. in the left eye. The palpebral fissure measured 4 mm. in width in the right eye and 9 mm. in the left eye.

There was restricted activity of the external and the superior rectus muscle and of the inferior oblique muscle on the right side.

Diplopia increased in the fields of action of the external rectus, the superior rectus and the inferior muscles of the right eye.

Anesthesia was present over the right malar area.

Roentgenographic Report (fig. 5).—Stereoscopic roentgenograms of the orbits showed that the margins were symmetric and without evidence of solution of continuity. There was complete destruction of the floor of the right orbit, with absence of visible fragments. The underlying antrum was densely clouded, and the soft tissue shadow suggested the prolapse of the orbital content into it. The lamina papyracea showed deformity, with compression of the ethmoid labyrinth and adjacent structures.

The impression was that of complete fracture of the floor of the right orbit and nasal wall with prolapse of orbital structures into the antrum.

Fig. 5 (case 3).—Roentgenogram of orbits, showing fragments of the floor and the orbital content filling the right antrum. The orbital margin and the adjacent structures are unaffected.

TREATMENT

Most of the patients with enophthalmos did not experience concomitant symptoms, and measures for correction of the displacement of the eye were not required. Indeed, several patients were unaware that the deformity existed. Several were much more interested in securing relief of diplopia than in having the eye restored to its normal position. For these patients measures to correct the diplopia were sufficient and gratifying.

Treatment to correct the deformity is receiving attention. Many procedures may be conceived for the reconstruction of the floor of the orbit and the restoration of the eye to its proper position. The use of autogenous tissue, bone or cartilage to form a new floor or of fat to fill the antrum, and thus raise the eye and orbital tissues, seems most likely to succeed. To fashion a new floor of viable tissue certainly requires consummate skill to overcome the technical difficulties. Filling of the defect in the floor of the orbit with fat taken from the abdominal wall is a relatively simple procedure but may not prove permanent. The use of preserved fascia lata in 1 case failed, for it acted as foreign material and had to be removed.

SUMMARY

Patients exhibiting signs of recent injury, such as the ecchymosis and edema of a "black eye," should have a roentgenographic examination for possible internal fracture of the orbit. The development of enophthalmos after trauma is always an indication of fracture, usually of the floor and/or the nasal wall, frequently without solution of continuity of other parts of the orbit. The posterior displacement of the eye is due to the increase in the capacity of the orbit, which results from the fragmentation of the floor with prolapse of orbital content in some degree into the antrum, and to fracture of the lamina papyracea with compression of the ethmoid cells.

Treatment may not be needed if the enophthalmos is not severe. If diplopia is present, in cases of less serious displacement the simpler means of relief may be operation on the extraocular muscles. In cases of severe enophthalmos operation to raise the orbital structures from the antrum may be gratifying to the patient.

CONCLUSION

Enophthalmos following trauma is due to fracture of the orbit. Internal fracture of the orbit, usually of the floor, without involvement of the margin, is a frequent result of a blow on the eye and should be diagnosed by roentgenographic means.

635 West One Hundred and Sixty-Fifth Street.

RECURRING ATTACKS OF CONCOMITANT EXOTROPIA, EACH FOLLOWED BY TRANSIENT ESOTROPIA

MIGRAINE THE PROBABLE CAUSE

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The present case is reported because it may throw light on the convergence mechanism and because, so far as I have ascertained, no such case has previously been described. However, the literature on oculomotor anomalies is so voluminous that reports of even a considerable number of such cases could easily be overlooked. No doubt cases of the kind have been observed but not reported. In fact, a colleague described to me a case which, judging from his brief account of it, was closely similar. The condition is not described in textbooks.

REPORT OF A CASE

An unmarried brunet, a Jewess, first consulted me on April 2, 1936, at the age of 28, and I have seen her at frequent intervals ever since. She complained of attacks which had begun to occur about seven months before her first visit to me. During an attack "everything looked crooked, people's faces looked queer" and her eyes "looked funny" to her friends. As will be evident, these phenomena were dependent on attacks of pronounced exotropia followed by transient esotropia. Up to the present she has had about twenty-four such attacks. Her refraction was as follows: In the right eye vision was 20/40, and with a correction of —0.50 D. sph. ⌒ —0.50 D. cyl., axis 105 it was 20/20—; in the left eye vision was 20/40, and with a correction of —0.75 D. sph. ⌒ —0.37 D. cyl., axis 90 it was 20/20—.

Between attacks, she had orthophoria within 2 prism diopters as indicated by the objective cover and the Maddox rod test. Hyperphoria never exceeded 1 prism diopter. Ocular movements were normal in all directions. She could overcome a prism of 14 D. base out and could not overcome one of more than 6 D., and sometimes not one of more than 2 D., base in. The near point of convergence was at 6 cm. With my new test, her acuity of stereopsis was 20/30. Her fundi and visual fields were normal. Her pupils were equal and round; they measured 2.5 to 3 mm. in diameter and reacted well to light but poorly in convergence. Her irises were dark brown.

Thus far the ocular attacks have consisted of two series separated by an interval of three and a half years. The first series covered a period of about one year, consisted of at least seven attacks and ended about Aug. 1, 1936. The second series, consisting of seventeen attacks, covered a period of one and a half years, beginning July 14, 1940 and ending about Dec. 1, 1941. Since then she had had no attacks, an interval of one and a half years at the time of writing. The interval between two attacks was often very short, the shortest being about one week.

I have never had the opportunity of examining her daily throughout an attack but have seen her several times during many of her attacks. I have seen her in every phase of an attack and frequently also in the intervals. According to her statements and my own observations, the attacks were all essentially alike. Three hours after she thought an attack had begun, exotropia of 40 prism diopters had developed in the left eye. The next day the exotropia was still 40 prism diopters. On the third day it was 30, and on the fourth, 20 prism diopters. Even when the exotropia was 40 prism diopters, she frequently overcame it and obtained bifixation, but only with the aid of excessive accommodation, which reduced her distance vision to 20/200. During fixation her visual acuity could be restored by the addition of a —1.50 D. sphere to her usual correction. For near range she had clear vision and bifixation without this addition. Her near point of convergence was at about 15 cm. The exotropia was as constant and as closely concomitant as that in an ordinary case of concomitant strabismus, although there was slight limitation of inward motion for each eye. When the

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exotropia had become 15 prism diopters, bifixation reduced the visual acuity to 20/50 —. With exotropia of 30 prism diopters and bifixation, it required fifteen seconds for vision to clear when one eye was covered. When the eye was uncovered, bifixation with blurred vision was achieved somewhat more quickly.

About one week after the onset of an attack, a stage was reached in which, during the examination, she sometimes had about 4 prism diopters of exophoria, and sometimes 4 prism diopters of esophoria. On one occasion, while she was being tested, she at first had orthophoria and then, for a few moments, as much as 8 prism diopters of esotropia. This stage I shall call the interphase of an attack. After this, relatively constant left esotropia developed, with diplopia, which disappeared in about a week. Sometimes a slight amount of esophoria persisted for a week longer. The greatest amount of esotropia I observed was 15 prism diopters. With this there was slight limitation of outward motion of each eye, and the convergence near point was at 3.75 cm. On one occasion diplopia was found to begin at a distance of 61 cm. There was never diplopia at the reading distance. In two attacks, with esotropia of 15 prism diopters, I found ortholaterophoria at 33 cm. with the vertical diplopia test.

In eight attacks I observed transition from exotropia to esotropia and I therefore assume that it occurred in all attacks. However, the patient thought that the first attack of the second series began with diplopia, and when I saw her on the following day she had esotropia of 8 prism diopters. During the exophoric phase she never observed diplopia, even when there was persistent exotropia. She also did not notice diplopia in the esophoric phase until I elicited it in my office. After this, she always observed diplopia when there was esotropia. She learned to distinguish the two phases by the fact that in the exophoric phase vision became blurred when things did not "look crooked" and that in the esophoric phase she had diplopia and distinct vision. Near vision, she thought, was never disturbed in either phase.

Although I proved to her that she could relieve her ocular discomfort at any time by occluding one eye, I could not induce her to employ this procedure because she thought it might cause her to "lose her job." I prescribed glasses containing an addition of a —1.75 D. sphere to her usual correction, to be used during the exophoric phase, but she seldom used them, although they gave her clear vision for distance.

With either eye occluded, distance vision was always the same, and the range of accommodation was 7 D. Hence in neither phase of an attack was there paresis or spasm of accommodation. The pupils during the attacks remained about the same size as before and responded well to light, and during the exophoric phase they responded as usual in convergence. During the esophoric phase their response in convergence was diminished, probably because then less effort was needed for convergence. At no time was nystagmus, mydriasis or definite miosis observed.

As a rule, hyperphoria was not revealed by the Maddox rod test during an attack. However, during several attacks she had left hyperphoria of 1 to 2 prism diopters. In one attack I found, with exophoria of 30 prism diopters, left hyperphoria of 8 prism diopters by the objective cover test. At or near the end of the esophoric phase of one attack, I once noted ortholaterophoria and 3 prism diopters of left hyperphoria with the Maddox rod test.

The patient stated that just before and during an attack she had a feeling of extreme fatigue. At or near the onset she had headache and nausea, and in some of the later attacks she vomited. The headache began over the left eye and spread to the upper jaw and then to the region of the left mastoid and left side of the occiput. She complained that there was sometimes a "terrific weight on the back of [her] neck, drawing [her] down." The headache did not prevent sleep and persisted two to four days. As a rule, its severity had greatly abated by the time the interphase was reached, and during the esophoric phase she did not complain of the pain.

She began to have headaches about ten years before I first saw her, and for the first few years they were not severe. Those associated with strabismus seemed to bear no constant relation to the menstrual periods, but the others occurred almost exclusively at these periods. She had never had scotoma of scintillating or any other type. At the beginning of a headache she frequently had a feeling of irritation as if from a foreign body in the left eye. With a severe headache she sometimes complained of peculiar feelings in one or the other arm. Shortly after the second series of ocular attacks ceased, she had a few attacks in which the "cords in the shoulder swelled," associated with a peculiar feeling in her throat and momentary difficulty in talking and swallowing. During the interval of three and a half years in which she had no ocular attacks, she had many attacks of headache with nausea, and such spells have been frequent during the present interval.

The patient is the fourth of 5 children; the others are all males. The second brother has frequent attacks of severe migraine associated with nausea. Another brother has alopecia areata. Her father recently died of a heart attack. Her mother, who is still living, has

occasional headaches. The patient remembers no illness except shingles. Her general physical condition is excellent. Her bowel movements and her menstrual periods are normal. She is moderately obese (height 5½ feet [167.6 cm.]; weight 167 pounds [75.7 Kg.]). On March 4, 1940 examination at the Lahey Clinic revealed nothing of importance. A roentgenographic examination of the gastrointestinal tract was made there. On May 28, 1941 I referred her to the Massachusetts General Hospital, where a thorough physical examination showed nothing abnormal. A Hinton test gave a negative reaction. The basal metabolic rate was + 4 per cent.

On Nov. 27, 1940 I referred her to Dr. G. Colket Caner for a neurologic examination. Aside from the ocular condition, his findings were normal. While she was in the esophoric phase of an attack he subjected her, at my suggestion, to a prostigmine test. His report follows:

"After subcutaneous injection of 2 cc. of a 1:2,000 solution of prostigmine methylsulfate and 1/100 grain (0.6 mg.) of atropine sulfate, the patient complained that she was not able to swallow, became extremely apprehensive and presented multiple peculiar somatic symptoms. Probably the feeling that she could not swallow was due to a spasm of her pharynx or esophagus, for twitching soon developed in all muscles, an occasional reaction in normal persons after injection of prostigmine.

"In addition to what seemed an hysterical reaction to the muscle spasms and twitchings produced by the prostigmine, the patient reacted in a neurotic way to the pain of the injection itself. In spite of all this, however, it is hard for me to believe that the ocular symptoms have a psychic origin. The result of the prostigmine test rules out myasthenia gravis." I may add that the injection had no effect on the esotropia.

In October 1941 I referred the patient to Dr. William G. Lennox for examination, with special reference to epilepsy and migraine. Excerpts from his reports follow:

"In addition to the major symptoms (headache, migraine, strabismus), she had a sensation of her nose being blocked and of a 'cool chill blowing through [her] face.' Presumably, this was paresthesia. There was also sometimes a 'dead feeling' in one arm, a sensation as though the 'muscles were not right.' This numbness might occur in either arm. There was also some trouble with her leg, which she failed to describe accurately. She did not complain of such sympathetic symptoms as flushing, chilliness and desire to urinate. She had taken no medication because of a prejudice against it, which was familial.

"The patient resembled her father in having a driving temperament. She was ambitious, conscientious, sensitive and easily upset emotionally, although her external appearance was placid. Although I did not go deeply into this aspect, she had the characteristics which are described as making up the migraine personality.

"The electroencephalogram was entirely normal. This is consistent with other cases of migraine in my experience, in which there is no personal or family evidence of epilepsy. The dead feeling which she had in her arm suggested a more extensive pathophysiologic condition.

"The patient let one of her full blown attacks pass without notifying me, but she came in the other day when she was having mild pain in the eye and symptoms suggestive of approaching strabismus. Her electroencephalogram was again entirely normal.

"I gave her 0.5 mg. of ergonovine subcutaneously and after an hour 0.4 mg. of ergonovine malleate intravenously. This treatment did not seem to influence the pain. Therefore, one must say either that she is one of the 10 per cent of patients with migraine who are not helped by ergot or that her headaches are not of the migraine type. The test was not fully conclusive because she was not having a full blown attack."

As possible causes of the ocular attacks in this case, neoplasm, multiple sclerosis, syphilis and various infectious processes are excluded by the frequency and the relatively short duration of the attacks and, above all, by the fact that in the seven and a half years since their onset no other neurologic symptoms have developed. Myasthenia gravis was excluded by the result of the prostigmine test. Hysteria seems to be eliminated by the presence of orthophoria between the attacks, and by the fact that without exophoria no one can voluntarily diverge the eyes. Moreover, the patient was not more emotionally unstable than most women of her age and race who are subject to migraine, and the attacks were not preceded by emotional distress. Epilepsy, remotely possible as a cause, was excluded by the electroencephalograms.

The only reasonable cause of the attacks seems to be migraine. Since this disease is conspicuously hereditary, it is of considerable significance that a brother of the patient was subject to frequent attacks of severe migraine. That another

brother had alopecia areata may also be of importance. But most significant is the fact that at the onset of each attack the patient had unilateral headache and other symptoms consistent with migraine and unexplained by any other cause.

The question now arises as to how migraine could produce such ocular attacks. In rare cases migraine causes transient paresis of one ocular nerve, almost always the third. The attacks may not always be associated with headache, and repeated attacks may result in permanent paresis of the affected muscles. In the present case, however, the ocular deviation was not paretic, and certainly was not dependent on involvement of one nerve alone. There are worthy of consideration, it seems to me, only three possible ways in which migraine could have caused the exophoric phase of the attacks in this case.

1. A vasomotor disturbance in the region of the nuclei of the sixth nerves might have caused hypertonia of these nuclei. Presumably, this would cause inhibition of the internal rectus muscles. As a result, exophoria, with some limitation of inward motion, would be expected. Concomitance of conjugate motion probably would not be greatly affected. The near point of convergence would no doubt recede, but to just how great an extent cannot be predicted because nothing is definitely known as to what determines the limit of normal convergence.

2. A vasomotor disturbance, probably vasoconstriction, might have reduced the tonus of the nuclei supplying both internal rectus muscles. Presumably, this would reduce the inhibition of the external rectus muscles and produce results comparable to those mentioned in 1.

3. A vasomotor disturbance might have affected the convergent mechanism. Little is known about this mechanism, but the evidence for Perlia's nucleus as a convergence center is strong. On the other hand, the evidence for a divergence center is weak. It consists chiefly of the occurrence of a condition known as divergent paralysis. However, one can explain this condition more satisfactorily, it seems to me, by not assuming the existence of a divergence center. But even if the existence of such a center is assumed, the fact that convergence was so slightly limited would seem to exclude divergence spasm as the cause of the exophoric phase in the present case. When bifixation has been in disuse for a considerable period, concomitant exotropia, often of high degree, develops. It does so despite the fact that to some extent stimulation to convergence continues through its association with accommodation. Presumably, therefore, the convergence mechanism contributes to the internal rectus muscle a tonus removal of which, together with the corresponding inhibition of the external rectus muscle, permits the eyes to diverge. Hence it seems probable that a vasomotor disturbance in the region of Perlia's nucleus could reduce its tonus sufficiently to produce concomitant exotropia of 40 prism diopters. Whether or not sufficient convergence innervation could be sent from the cortex to restore this tonus and, in addition, make possible convergence of the eyes to 15 cm. is problematic. However, it seems probable, especially if, as I believe, the limit of convergence is normally imposed by the degree of inhibition of the antagonists. Moreover, in many cases of exotropia of 40 prism diopters the patient can converge his eyes to 15 cm.

The second of the aforementioned possible causes for the exophoric phase of the attacks would seem to be more probable than the first because migraine has seldom been known to affect even one external rectus muscle and because, so far as has been observed, it depresses the activity of the ocular muscles it affects. The third possibility seems the most probable of the three because the vasomotor disturbance would need not only to be depressive but to involve a smaller area and would affect both eyes equally.

On the basis of the preceding explanation for the exophoric phase the esophoric phase is easily accounted for, which makes the whole explanation still more probable. The esophoric phase cannot have been caused by so-called divergence paralysis, because in this phase the near point of convergence was brought nearer to, not farther from, the eyes. It could be explained on the assumption that a convergence spasm resulted from the great effort required to overcome the preceding exotropia, or on the assumption that an excitatory state in Perlia's nucleus occurred as a rebound from the state of depression. The second assumption seems the more probable because the esotropia was not accompanied by miosis or by spasm of accommodation. It is to be noted that this explanation of the esophoric phase accords with the fact that the esotropia was always far less severe than the preceding exotropia. The instability of the muscle balance in the interphase is satisfactorily explained by the transition from the assumed depressed to the excitatory states in Perlia's nucleus.

Possibly the conditions described would result from a vasomotor disturbance in the region of a cortical center for convergence. The facts, however, that Perlia's nucleus is practically continuous with the nuclei of the third nerve and that ophthalmoplegic migraine almost exclusively involves the third nerve make it more reasonable to assume that it was Perlia's nucleus which was affected in the present case. If it was true, as the patient stated, that in one attack esotropia was not preceded by exotropia, it would seem that migraine can cause hypertonus of Perlia's nucleus without first depressing it. If future investigations should prove that Perlia's nucleus is not part of the convergence mechanism, it would become necessary to assume that some unknown region concerned with convergence was affected in the present case.

I assume that both the sensory and the motor phenomena associated with migraine are due to localized vasomotor disturbances in the brain. That the sensory phenomena have this cause I have not the slightest doubt. Recently I examined sections of the occipital lobe of 1 of my patients, subject to migrainous scintillating scotoma, who thirty-one years before his death had suddenly acquired a permanent peripheral scotoma, similar in the two eyes. The sections showed a localized lesion which evidently was produced originally by necrosis and which, so far as could be determined, corresponded to the scotoma. Other explanations have been advanced for the oculomotor paresis of migraine, notably, pressure on the affected nerve by a temporarily swollen hypophysis or by a temporarily distended large vessel. These explanations seem unlikely in view of the fact that they are not applicable to the sensory phenomena. The present case makes them still more improbable, since the lesion in this instance could not have been peripheral.

SUMMARY AND CONCLUSIONS

In the case reported there occurred periodically nonparetic severe exotropia or exophoria, followed by transient esotropia. The exophoric and the esophoric phase of an attack each persisted about one week. At the onset of each attack the patient had severe headache, nausea and other symptoms consistent with migraine and not explained by any other cause.

It is safe to conclude that these remarkable oculomotor phenomena were produced by a recurring disturbance situated not farther peripheral than the midbrain and dependent on migraine, not hysteria.

The attacks seem best to be explained by the assumption that a vasomotor disturbance associated with migraine first depressed and then temporarily raised the tonus of Perlia's nucleus.

395 Commonwealth Avenue.

KERATOCONUS POSTICUS CIRCUMSCRIPTUS

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Among the rare forms of corneal abnormalities, there exists a group characterized by anomalies of curvature of the posterior corneal surface. Butler has named and described two types of abnormal posterior corneal curvature. One form is called keratoconus posticus and is characterized by a perfectly regular and unusually short radius of curvature for the entire posterior corneal surface. The other form, called keratoconus posticus circumscriptus, shows a localized area of increased curvature on the posterior corneal surface. In both types the anterior corneal curvature is smooth, regular and of average radius. In both types the cornea is thinned in the area of increased curvature. This thinning involves the entire cornea in the first, or keratoconus posticus, type and is limited to a circumscribed area in the second type.[1]

Stallard,[2] in 1930, reported the first case of so-called keratoconus posticus circumscriptus. Butler [2b] and Ingram [3] each recorded a case. All these reports have appeared in the English literature; evidently no case has been recorded in the American journals.

REPORT OF A CASE

A Negress aged 36 reported to the ophthalmologic clinic of the Hospital of the University of Pennsylvania, desiring a refraction, having recently broken her glasses. As far as she recalled, there had never been any injury to or inflammation of either of her eyes. However, she has always been aware of poorer vision in her left eye. She had been wearing glasses for ten years. Visual acuity without correction was 6/9 + 4 in the right eye and 6/60 in the left eye; with the best correction after cycloplegic examination it was 6/5 in the right eye and 6/30 in the left eye. External examination revealed nothing unusual concerning the lids, fissure or conjunctiva of either eye. The cornea of the right eye was clear and sensitive, with no irregularities, staining or opacities. The cornea of the left eye showed a slight, circumscribed haze at approximately 7 o'clock, just below the central area. The corneal reflex was present to a wisp of cotton, and the cornea did not stain with fluorescein. Intraocular tension was 20 mm. of mercury in each eye, measured with a Schiøtz tonometer. The pupils were equal and regular. They reacted equally and promptly, with good amplitude, to direct and indirect stimulation with light and to a near stimulus. Ocular movements were full, both for disjunctive and for conjugate movements. The cover test revealed only slight exophoria for near vision and orthophoria for distance. The lacrimal sacs were both free from discharge, and the nasolacrimal ducts were patent.

Slit lamp examination of the right eye revealed nothing pathologic. Examination of the left eye by sclerotic scatter (figure *A*) revealed a circumscribed, horizontally oval haze lying nasally just below the central area of the cornea. Most of the opacity as seen in optical section (figure *B*) lay in the posterior portion, and a definite thinning existed in this area. The cornea here was approximately one-half the thickness of the rest of the cornea. The anterior surface of the cornea was smooth and possessed the same curvature throughout, with no irregularity in the area of the lesion. The posterior radius of curvature, in the area of the lesion, was notably shortened. This curvature was not regular, but was most pronounced in

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1. Butler, T. H.: (*a*) Keratoconus Posticus, Tr. Ophth. Soc. U. Kingdom **50**:551, 1930; (*b*) Two Rare Corneal Conditions, Brit. J. Ophth. **16**:31, 1932; (*c*) Discussion of the Rarer Forms of Keratitis, Tr. Ophth. Soc. U. Kingdom **57**:32, 1937.

2. Stallard, in discussion on Butler.[1a]

3. Ingram, H. V.: Keratoconus Posticus, Tr. Ophth. Soc. U. Kingdom **56**:263, 1936.

the lower portion. No pigmentation could be seen on the posterior corneal surface, nor was an aqueous flare seen. Both the lens and the iris failed to reveal any signs of trauma or disease.

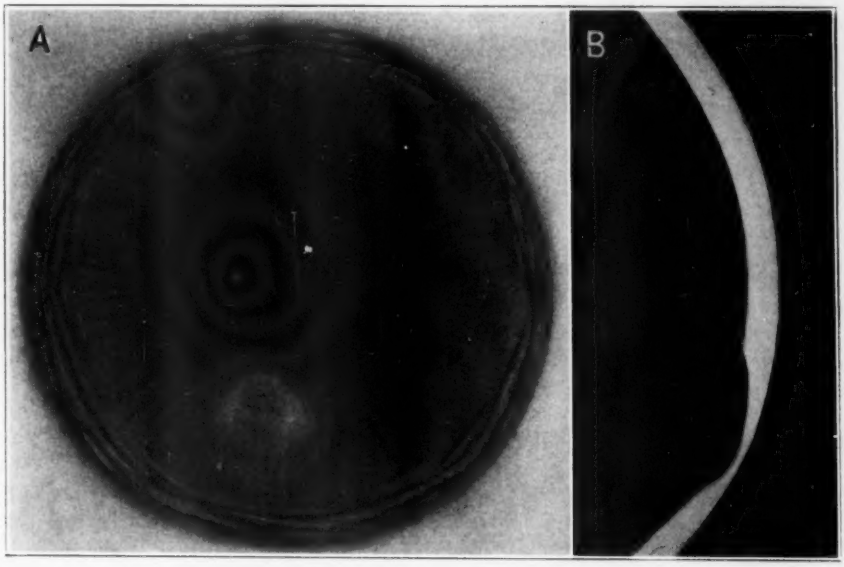
After instillation of 8 drops of 2 per cent homatropine hydrobromide, refraction with post-cycloplegic examination revealed 6/5 vision in the right eye, with a correction of +25 D. sph. ⊃ +25 D. cyl., axis 160, and 6/30 vision in the left eye, with a correction of +2.00 D. cyl., axis 5. A pinhole disk failed to improve vision in the left eye.

Ophthalmoscopic examination revealed no pathologic process, although the view of the left fundus was slightly hazy, as is usual in an astigmatic eye. Inspection of the visual fields revealed no scotomas.

Placido's rings appeared circular, and keratometric reflections were regular.

COMMENT

The signs observed in this case entitle the condition to be classed as a type of circumscribed posterior keratoconus. The failure to obtain improved vision in



A, corneal haze, as seen by sclerotic scatter; *B*, optical section, showing increased posterior corneal curvature in the region of corneal haze, with a normal anterior curvature.

Summary of Reported Cases of Keratoconus Posticus Circumscriptus

Observer	History of Injury	Age, Yr.	Visual Acuity	Refractive Error	Position of Lesion	Sex
Stallard.....	+	Middle-aged	6/24	.....	Central	M
Butler.....	+	29	6/12 (1930) 6/ 9 (1931)	+2 D. cyl., axis 30 —2 D. cyl., axis 180	Not quite central	M
Ingram.....	—	54	6/60	Could not be improved	Central	F
Present case.....	—	36	6/30	+2 D. cyl., axis 5	Not quite central	F

the left eye may be the result of amblyopia ex anopsia following uncorrected anisometropia of childhood.

The cases reported in the literature are indicated in the accompanying table.

In 2 of the cases reported there was a history of injury and in 2 there was none. No one actually saw this condition develop after an injury in a healthy cornea. So, at best, the assumption of a traumatic origin is *post hoc ergo propter hoc* reasoning and may suffer the fallacies of all such logic.

SUMMARY

A case of keratoconus posticus circumscriptus is presented. No history of preceding disease or injury could be elicited.

CLASSIFICATION OF EXPERIMENTAL CATARACTS IN THE RAT

RECENT OBSERVATIONS ON CATARACT ASSOCIATED WITH TRYPTOPHAN DEFICIENCY AND WITH SOME OTHER EXPERIMENTAL CONDITIONS

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Since the early observations by Erdheim,[1] in 1906, on cataract associated with tetany in rats and those by A. Buschke,[2] in 1913, on cataract produced by thallium, cataract has been observed with seven other experimental conditions of the rat. Such cataracts do not include the acute reversible opacities of the lens due to osmotic and physical factors. The question arises whether this fairly large body of experimental observations can be put to any use in solution of the problem of the pathogenesis of cataract. The occurrence of cataract in one species under a great number of different conditions appears to be a fortunate situation from the standpoint of comparative pathology.

Valuable information can be obtained by systematic studies with the slit lamp during the early stages and throughout the development of the cataract and by observation on allied manifestations elsewhere in the body. On the basis of such observations, Goldmann [3] differentiated thallium cataract and tetanic cataract and showed that the former cannot be explained by an insufficiency of the parathyroids. Observations on the allied symptoms of galactose cataract have led other authors [4] to suspect the relation of this cataract to osmotic disturbances. Similar observations, which include studies of the other experimental cataracts in the rat, permit a

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This work was supported in part by grants for the study of amino acids in nutrition made by the Rockefeller Foundation, Merck & Company, Inc., Eli Lilly and Company, and E. R. Squibb & Sons to the Department of Pediatrics, Johns Hopkins University School of Medicine. The work on riboflavin deficiency and on galactose and xylose cataracts, briefly referred to in this paper, is being carried on under grants from the John and Mary R. Markle Foundation.

The observations on tryptophan-deficient rats reported in this paper are part of a general study on the clinical and chemical changes occurring in amino acid deficiencies in experimental animals and in human beings. This study was conducted in the department of pediatrics of the Johns Hopkins Hospital and University and was under the immediate direction of Dr. L. Emmett Holt Jr. The participation of the department of ophthalmology in the study was incidental and was made at the specific invitation of Dr. Holt. Full reports of the study as a whole will be made by Dr. Holt and his co-workers in other journals. The observations reported here are, however, of special ophthalmologic interest and on this account appear to warrant a separate report in the ophthalmologic literature.

The cataracts in rats with experimental diabetes occurred in the course of studies conducted by Dr. Curt P. Richter in the psychobiologic laboratory of the Phipps Psychiatric Clinic, Johns Hopkins Hospital, who also observed the cataracts. Dr. Richter permitted me to study these eyes. Reference to these studies is made by permission of Dr. Richter.

1. Erdheim, J.: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **16**:632, 1906.

2. Buschke, A.: Arch. f. Dermat. u. Syph. **116**:477, 1913.

3. Goldmann, H.: Arch. f. Ophth. **122**:146, 1928.

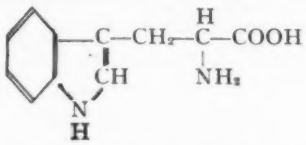
4. (*a*) Süllmann, H., and Weekers, R.: Ztschr. f. Augenh. **95**:58, 1938. (*b*) Bellows, J. G., and Chinn, H.: Theories of Cataract, Arch. Ophth. **26**:1066 (June) 1941.

classification of these cataracts into three fairly well circumscribed groups; the diabetic, the tetanic and the dystrophic.

In the present study, in addition to the cataracts associated with tryptophan deficiency and with experimental diabetes, some observations have been made on riboflavin-deficient rats and on the cataracts due to galactose and xylose. As a result of these comparative studies, a classification of experimental cataracts is made, with the object of delimiting the individual groups of cataract.

OBSERVATIONS ON TRYPTOPHAN-DEFICIENT RATS

Tryptophan is an indole aminopropionic acid and has the following structure:



It can be replaced in the diet only by a few other indole compounds, e. g., indole pyruvic acid. It has been known for many years that the absence of tryptophan from the diet results in the arrest of body growth of young animals.[5] In addition, some specific manifestations of the deficiency have been observed. In rats with experimental tryptophan deficiency, blindness with opacities in the ocular media was noted by Curtis, Hauge and Kraybill,[6] and the occurrence of cataract and vascularization of the cornea in this condition was reported by Totter and Day.[7]

In the present study, attention has been especially directed toward the morphologic character and mode of development of tryptophan deficiency cataract and its association with lesions in certain other organs.

MATERIAL AND METHODS

Both albino and hooded rats were used by Dr. Holt and Dr. A. A. Albanese in their experiments. The rats belonged to two strains, of which one was bred as a mixed strain in the laboratory of the department of pediatrics and the other was an unidentified strain and had been obtained by Dr. Albanese from an out-of-state source.

The dietary study was carried out as a "paired feeding" experiment; i. e., the food intake of the control animals was restricted to that of the deficient animals of the same age in order to rule out inanition as a possible cause of symptoms. The diet used by Dr. Holt and Dr. Albanese was compounded as follows: protein (acid-hydrolyzed casein concentrate), 147 Gm.; l-cystine, 1.5 Gm.; sucrose, 150 Gm.; starch, 420 Gm.; agar, 20 Gm.; salt mixture (see next paragraph), 20 Gm.; hydrogenated cottonseed oil (Crisco), 190 Gm.; brewers' yeast (Mead Johnson), 42.7 Gm.; Mead Johnson's cod liver oil substitute,[7a] 50 Gm., and water to make the proper consistency.

The salt mixture used had the following composition: sodium chloride, 18.9 Gm.; dicalcium phosphate, anhydrous, 25 Gm.; magnesium sulfate, anhydrous, 6.86 Gm.; potassium bicarbonate, 44.4 Gm.; potassium chloride, anhydrous, 2.88 Gm.; ferric citrate U. S. P., 2.21 Gm.; cupric sulfate, anhydrous, 0.24 Gm.; manganese sulfate, anhydrous, 0.15 Gm.; potassium iodide, 0.015 Gm., and sodium fluoride, 0.03 Gm.

The diet of the control animals was exactly like that of the deficient animals except for the supplementation with 2.25 Gm. of tryptophan per thousand grams of food.

In the first series of their experiment, in which were made most of the observations reported here, 8 animals of about 100 Gm. body weight were placed on the deficiency diet

5. Berg, C. P.; Rose, W. C., and Marvel, C. S.; J. Biol. Chem. **85**:207 and 219, 1929. Jackson, R. W.: ibid. **73**:523, 1927; **84**:1, 1929.

6. (*a*) Curtis, P. B.; Hauge, S. M., and Kraybill, H. R.: J. Nutrition **5**:503, 1932. (*b*) Berg, C. P., and Potgieter, cited by Totter and Day.[7]

7. Totter, J. R., and Day, P. L.: (*a*) J. Biol. Chem. **140**:cxxxiv, 1941; (*b*) J. Nutrition **24**:159, 1942.

7a. The substitute contains fish liver oil, sardine oil and maize oil plus viosterol.

and a corresponding number on the control diet. In addition, 4 adult albino rats of between 200 and 300 Gm. were placed on the deficiency diet.

In addition to this series, observations were made on the eyes of a larger series (over 30) of similarly deficient and control rats. These rats, however, were killed at various times during the experiment to provide material for the study of the systemic lesions. Systematic observations on the eyes of these animals were therefore not possible. In general, the cataractous and corneal changes observed were the same as those systematically studied in the first series. Reference will later occasionally be made to this larger series.

The animals were examined with the slit lamp about once a week, with the use of atropine mydriasis, and during the development of the cataracts they were examined every day.

For slit lamp observation the rats were examined on a stand, as shown in figure 1. For slit lamp drawings the rats were immobilized by subcutaneous injection of 0.25 cc. per hundred grams of body weight of a 1:10 dilution of a commercial bulbocapnine solution.[8]

TRYPTOPHAN DEFICIENCY CATARACT

This type of cataract has been observed only in young rats, never in adults. It occurs in two morphologically distinct forms, the acute and the chronic.

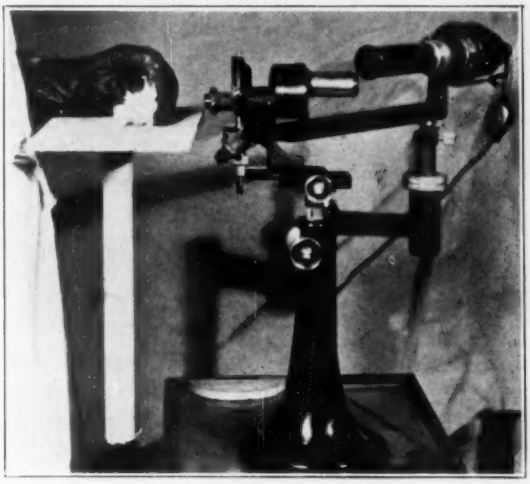


Fig. 1.—Slit lamp examination of the rat's eye.

Acute Type of Cataract.—The acute type was observed in 3 of 4 young animals, all of the same strain. The general pathologic picture was as follows:

Seven to eleven weeks after the animal was placed on the deficient diet, a feathery opacity, with leaflike margins, developed in the posterior subcapsular cortex. The suture lines were clearly visible and gaped slightly, and some medium-sized vacuoles were visible along the suture lines. The final opacity was somewhat denser at the peripheral margins than at the posterior pole of the lens (fig. 2 *A* and *B*).

While the opacities in the posterior cortex were developing, the anterior cortex and the nucleus remained clear. After the development of the posterior opacity short, spokelike opacities appeared in the anterior cortex near the equator, followed within four days by the development of a shell-like, perinuclear opacity. This consisted of innumerable fine motes, which gave a grayish, milky appearance to the anterior cortex (fig. 2 *C* and *D*). The nucleus now rapidly became opaque,

8. Richter, C. P., and Paterson, A. S.: J. Pharmacol. & Exper. Therap. **43**:677, 1931.

and the perinuclear opaque zone became wider. The anterior suture lines gaped, to become narrower again as the cataract became mature (fig. 2 *E* and *F*). The cataract became mature within three weeks after the onset of the first changes.

Chronic Type of Cataract.—This type of cataract was seen in 3 of 4 animals of one strain and in 1 of the other strain. The general pathologic picture was as follows:

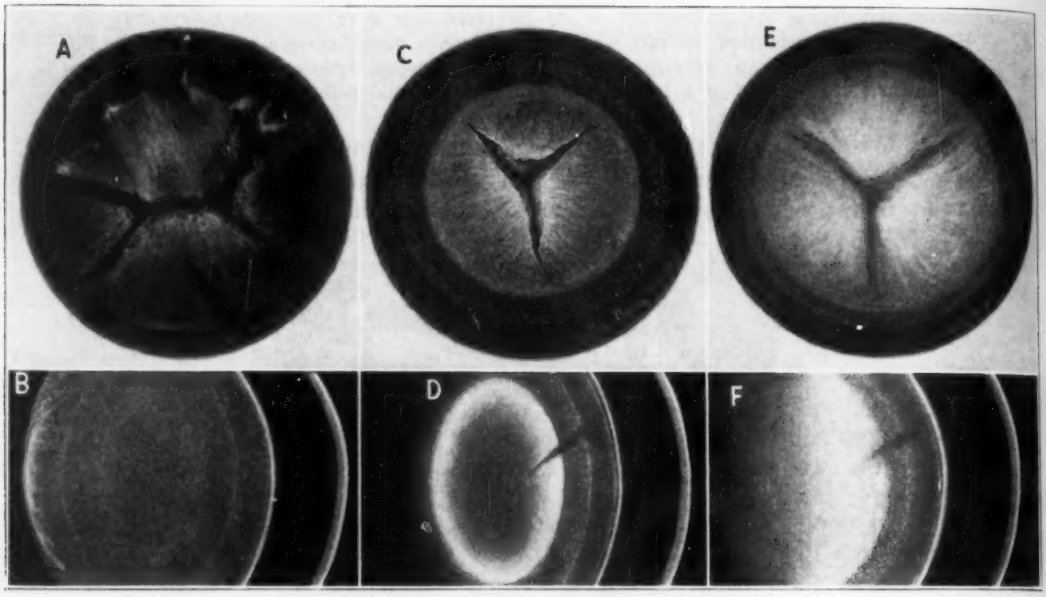


Fig. 2.—Cataract associated with acute tryptophan deficiency. *A*, composite drawing of the posterior cortex and, *B*, optical section in the first stage. *C*, composite drawing of the posterior cortex and, *D*, optical section in the second stage. *E*, composite drawing of the posterior cortex and, *F*, optical section in the third stage.

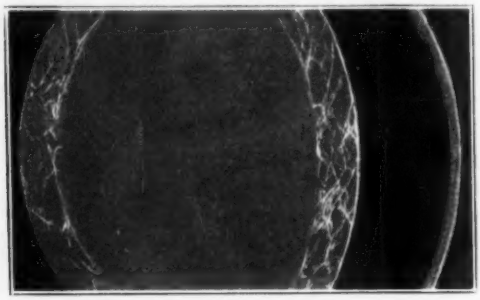


Fig. 3.—Optical section in a case of tryptophan deficiency cataract, chronic form, in the rat.

Eight to ten weeks after the rat was placed on the tryptophan-deficient diet, fuzzy opacities appeared along the anterior subcapsular suture lines, and the feathery structure of the superficial cortex became visible. Fine, dotted opacities appeared in the superficial portion of the cortex. During the following weeks a fine network of cobweb-like opacities developed in the anterior and the posterior superficial portions of the cortex (fig. 3), and this zone of opacities gradually

gained in depth. Occasionally a few fine vacuoles were visible in this zone. The deep cortex and the nucleus remained clear, and the cataract did not mature during the lifetime of the animal, i. e., within three to nine weeks after the onset of the first changes.

In 1 rat a combination of the acute and the chronic type of cataract was seen. In this animal cobweb-like opacities appeared in the superficial cortex, and a flat, dense, leaflike opacity developed around the posterior pole of the lens. Within six to eight weeks after the onset of the first changes, perinuclear and nuclear opacities developed in one eye, and the feathery arrangement of the fine superficial cortical opacities became clearly visible. The suture lines gaped. However, the cataract in the other eye did not progress beyond the stage of the posterior polar opacity (fig. 4).

Arrest of the Acute Cataract by Supplementation of the Diet with Tryptophan.—To 1 animal, tryptophan was supplied at the time that the leaflike opacity had developed in the posterior subcapsular region of the lens in one eye.

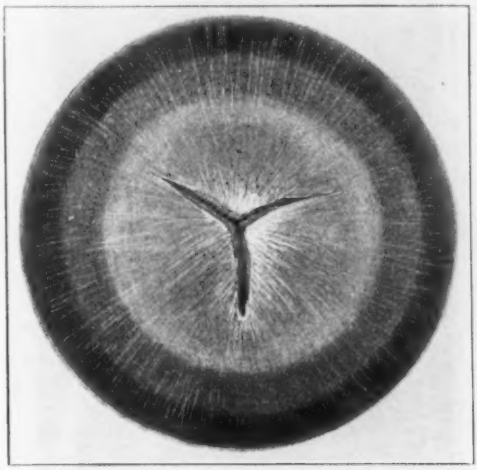


Fig. 4.—Composite drawing of tryptophan deficiency cataract, combined form, in the rat.

During the first five weeks after the supplementation of the diet, some snowflake opacities appeared around the nuclear suture lines, and in one eye a flat, rosette-like opacity developed at the anterior nuclear surface (fig. 5 *A*). However, the cataracts did not progress further, and even during these first five weeks after supplementation of the diet a clear subcapsular zone of fresh lens substance was deposited around the optically denser deep portion of the cortex. This clear superficial zone was separated from the deeper cortex by a dense zone of discontinuity (fig. 5 *B*). In the following months the clear superficial zone became gradually wider and produced the picture of a lens within a lens.

It is of interest that when tryptophan was administered to a deficient animal some further opacities of the lens developed even though the general condition of the rat had begun to improve and the animal had started to gain weight. It is evident that some latent, irreparable damage to the lens fibers had taken place already and led eventually to manifest opacities. Similar phenomena are well

known in the realm of injuries due to actinic rays and have been reported also in cases of tetanic [8] and of galactose cataract.[9]

Histologic Studies.—In the cataractous lens, globular degeneration of the lens fibers, particularly in the superficial cortex, was noted. In addition, pronounced proliferation of the epithelium was seen in the cataract complicating acute tryptophan deficiency.

Histochemical Tests for the Presence of Bound Indoles.—Tryptophan and other indoles form colored products in the presence of aldehydes. Tryptophan must be split off by hydrolysis from the protein molecule before it gives this reaction.

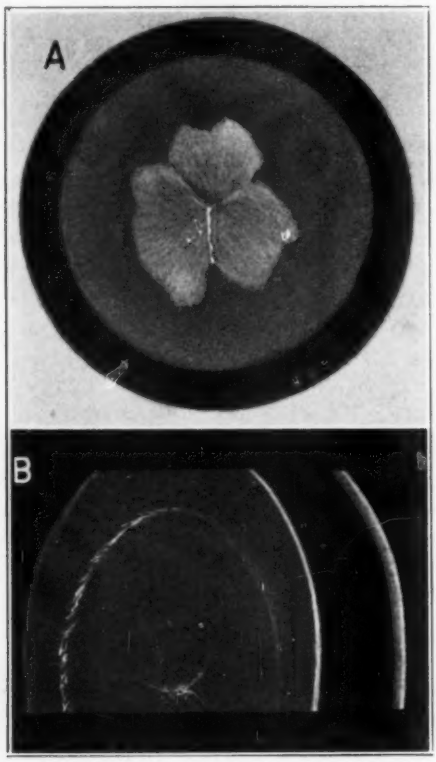


Fig. 5.—*A*, composite drawing, and, *B*, optical section (other lens), of tryptophan deficiency cataract arrested by supplementation of the diet with tryptophan.

The reagents employed in the test are (1) the vanillin reagent, consisting of a 1 per cent solution of vanillin in 50 per cent hydrochloric acid; (2) concentrated hydrochloric acid, and (3) 3 per cent hydrogen peroxide.

Method: Paraffin sections of material fixed in solution of formaldehyde U. S. P. are deparaffinized, carried through the alcohols to water and dried between filter paper. One drop of concentrated hydrochloric acid is placed on the section and left there about one-half minute. One drop of the vanillin reagent is added without the sections being washed, and one-half minute later 1 drop of 3 per cent hydrogen peroxide is added. After some minutes the fluid is drained off with filter paper, and the preparation is washed with distilled water. It can then be carried through the alcohols and xylene and mounted in Canada balsam. A qualitatively positive reaction is an intensely red color.

9. Darby, W. J., and Day, P. L.: J. Biol. Chem. **133**:505, 1940.

The histochemical test for bound indole, presumably tryptophan, in the cataractous lens of the tryptophan-deficient rat still gives a definitely positive reaction, even in the advanced stages of cataract. Sometimes the reaction is slightly weaker than that in the control lenses, particularly in the cortex of the lens, but it has never been negative. The result indicates that even in the advanced stages of tryptophan deficiency cataract no complete loss of bound indole from the proteins of the lens has taken place. This method, however, does not lend itself to quantitative conclusions.

Changes in the Lens in Control Animals.—The control animals were not maintained on a normal stock diet but received the same diet as the tryptophan-deficient animals supplemented only with tryptophan, and the quantity was restricted to the food intake of the deficient rats.

In some of the control rats there developed minor changes in the lens which deserve special description. The most important lesions were abnormally visible deep cortical zones of discontinuity or thin zonular opacities. The changes could not have been detected with an ophthalmoscope alone and were visible only with the slit lamp.

In order to find out whether these lesions might be strain-specific, endogenous changes in the lens, 31 rats of the same strain aged from 3 to about 6½ months which had been kept on unlimited stock diet were examined. Of these normal rats, only 1 showed a circumscribed reflecting zone in the deep cortex.

It is thus likely that the zonular changes seen in some of the rats on a tryptophan control diet did not represent a hereditary type of lesion but, rather, were due to some exogenous damage associated with the difference between the tryptophan control diet and the ordinary stock diet. The tryptophan control diet was not sufficiently inadequate in regard to quality and quantity to produce progressive cataract or damage to the lens of such magnitude that it could not be overcome during the later growth of the lens. However, the control diet may have exerted a transitory influence on the lens at an early period which was "outgrown" later by the normal growth of the lens from the periphery.

It is possible that the lens-damaging factor or factors intrinsic to the tryptophan control diet predisposed to manifestations of damage to the lens encountered in the rats on the tryptophan-deficient diet. It is not known which factor or factors these were. Some connection with the quantitative restriction of the diet, as used in the paired feeding technic, is possible.

LESIONS ASSOCIATED WITH TRYPTOPHAN DEFICIENCY CATARACT

Changes in the Cornea.—Vascularization of the cornea and keratitis were observed in tryptophan-deficient rats by Totter and Day.[7] In the tryptophan-deficient animals of Dr. Albanese and Dr. Holt, vascularization of the cornea was seen with the slit lamp in 6 of 8 of the young rats in the first series and in the majority of the rats of the second series. In some rats weighing 40 Gm. at the time they were placed on the deficient diet, vascularization was seen within ten days. It also occurred in adult rats receiving the deficient diet. In general, the vascularization of the cornea in the young rats was well advanced by the time the cataract developed. Narrowing of the corneal vessels to invisibility was observed on examination with the slit lamp within a few weeks after supplementation of the diet with tryptophan. None of the control animals in the paired feeding experiments showed this vascularization.

On examination with the slit lamp the vessels appeared as predominantly superficial loops originating in the superficial perilimbal plexus (fig. 6). Often fine, straight sprouts arose from the vertex of a capillary loop and ran in a radial direction. Under a high power lens circulation of the blood could be seen in the wider loops.

Study of flat preparations of the cornea after injection of india ink (method of Bessey and Wolbach [10]) showed, under a higher power lens, that the fine sprouts arising from larger capillary loops finally bent toward similar sprouts arising from another capillary loop (fig. 7). Obviously, new capillary loops are formed in this way.

Infiltration of the cornea was seen in some eyes of tryptophan-deficient rats. It began sometimes around the vertex of capillary loops, and in some cases the whole cornea became cloudy and "steamy." Occasionally, in advanced stages the surface of the cornea appeared dry.

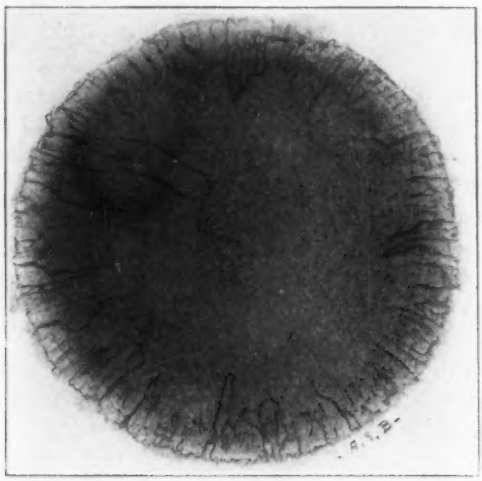


Fig. 6.—Vascularization of the cornea associated with tryptophan deficiency in the rat. Composite drawing with the slit lamp.

Histologic sections confirmed the predominantly superficial location of the capillaries (fig. 8 *A*); sometimes the epithelium was even elevated. In more advanced stages capillaries were also visible in the deeper layers of the stroma, together with polymorphonuclear and round cell infiltration (fig. 8 *B*).

For comparison, I have studied the corneal lesions associated with riboflavin deficiency.

The diet of the riboflavin-deficient rats was made up of Smaco, a vitamin B complex-free basal diet,[10a] with the following supplements in the amounts stated per kilogram of basal diet: thiamine hydrochloride, 8 mg.; pyridoxine hydrochloride, 12 mg.; calcium pantothenate, 500 mg.; choline chloride, 2,000 mg.; nicotinic acid, 200 mg., and inositol, 200 mg. The diet of the control animals contained, in addition, 12 mg. of riboflavin per kilogram of basal diet.

The corneas were examined with the slit lamp, in flat preparations after injection of india ink and in histologic sections. On the basis of these studies, I can

10. Bessey, C. A., and Wolbach, S. B.: J. Exper. Med. **69**:1, 1939.

10a. The composition of the vitamin B complex–free diet was as follows: sucrose, 68 per cent; vitamin-free casein, 18 per cent; vegetable oil, 8 per cent; salt mixture no. 2 U. S. P., 4 per cent, and cod liver oil, 2 per cent.

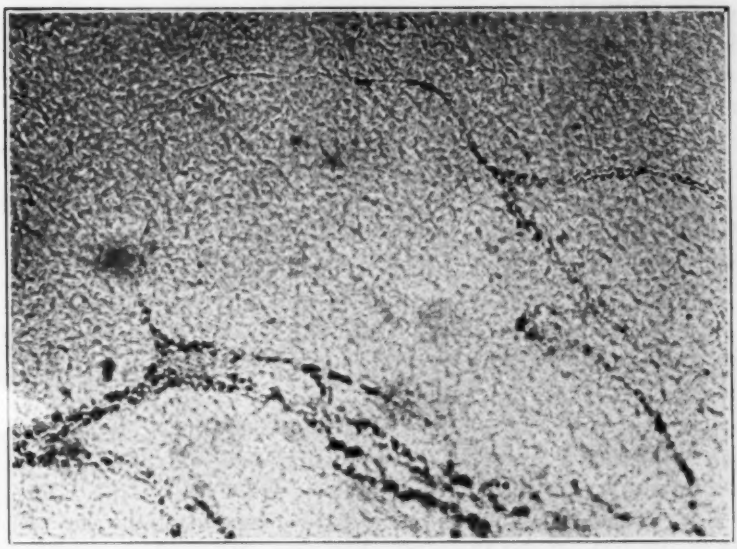


Fig. 7.—Photomicrograph showing vascularization of the cornea associated with tryptophan deficiency in the rat. Injection with india ink.

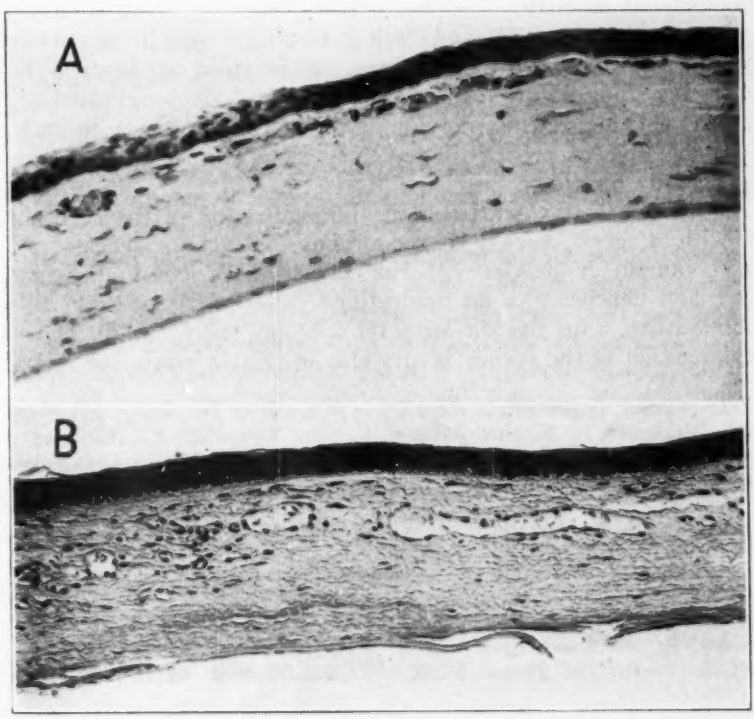


Fig. 8.—Photomicrographs showing corneal changes associated with tryptophan deficiency in the rat. *A*, subepithelial vascularization; *B*, vascularization and round cell infiltration. Hematoxylin and eosin stain.

amply confirm the descriptions of earlier observers,[11] but, contrary to the reports of Totter and Day,[7] I was unable to note any significant differences between the corneal lesions in the riboflavin-deficient and those in the tryptophan-deficient animals.

Arrest of Body Growth in Young Animals.[5]

Alopecia and Other Cutaneous Lesions.[12]—These changes occurred both in young and in adult rats. Totter and Day,[7b] using two different diets, observed cutaneous changes only with one of the diets. Predisposing dietary factors other than tryptophan deficiency may play a role here, too.

Atrophy of the Testicles and Aspermatogenesis.[13]

Lesions in the Incisor Teeth.[14]—It is to be noted that the rat's incisor teeth, like the lens, grow throughout life. The relation of lesions in these teeth to tryptophan deficiency, however, demands further study.

COMMENT

Relation of Ocular Lesions to Tryptophan Deficiency.—Whether or not any of the manifestations are secondary to lesions elsewhere in the body cannot be decided. It is certain that not all the observed manifestations can be secondary to changes in the testicles because castration does not produce similar symptoms.

A secondary inanition deficiency due to diminished food intake is unlikely because the experiment was carried out as a paired feeding experiment, and none of the control animals manifested any of the forms of cataract observed in the tryptophan-deficient animals.

Likewise, "cold cataract" [15] and "salt cataract," [16] which have been observed in very young rats on exposure of the eyes, can be ruled out because the cataracts described in this study occurred in rats of 100 Gm. body weight and the opacities were not reversible and did not develop as rapidly as those seen in such cataracts.

The possible role of predisposing dietary factors other than tryptophan deficiency has already been discussed.

It is of interest that vascularization and infiltration of the cornea have been observed with a great variety of conditions: riboflavin deficiency,[11] thallium poisoning,[17] vitamin A deficiency,[18] zinc deficiency,[19] sodium deficiency,[20] lysin deficiency [7b] and deficiency of an unidentified factor of the vitamin B complex.[21] Recent observations with the slit lamp on a highly inbred strain of rats suggest that vascularization of the cornea occurs also in certain strains of rats as a geno-

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11. O'Brien, C. S.: Experimental Cataract in Vitamin G Deficiency, Arch. Ophth. **8**:880 (Dec.) 1932. Eckhardt, R. E., and Johnson, L. V.: Nutritional Cataract and Relation of Galactose to Appearance of Senile Suture Line in Rats, ibid. **21**:315 (Feb.) 1939. Bessey and Wolbach.[10]

12. (*a*) Buschke, W.; Albanese, A. A., and Follis, R. H., Jr.: Federation Proc. **1**:175, 1942. (*b*) Albanese, A. A., and Buschke, W.: Science **95**:584, 1942. (*c*) Totter and Day.[7b]

13. Shettles, L. B.: Proc. Third Ann. Conf. Biol. Spermatozoa, 1942, p. 28.

14. Buschke, Albanese and Follis.[12a] Albanese and Buschke.[12b]

15. Goldschmidt, M.: Klin. Wchnschr. **6**:635, 1927.

16. Goldmann, H., and Rabinowitz, G.: Klin. Monatsbl. f. Augenh. **81**:771, 1928.

17. Ginsberg, S., and Buschke, A.: Klin. Monatsbl. f. Augenh. **71**:385, 1923.

18. Wolbach, S. B., and Howe, P. R.: J. Exper. Med. **42**:753, 1925. Bessey and Wolbach.[10]

19. Follis, R. H., Jr.; Day, H. G., and McCollum, E. V.: J. Nutrition **22**:223, 1941.

20. Follis, R. H., Jr.; Orent-Keiles, E., and McCollum, E. V.: Am. J. Path. **33**:504, 1942.

21. György, P.; Johnson, L. V., and Eckhardt, R. E., cited by Evans, E. A.: The Biological Action of the Vitamins, Chicago, University of Chicago Press, 1942, pp. 64-65.

typical trait when the animals are receiving normal diets. Occasionally superficial blood vessels were seen in histologic sections of the cornea of old rats on normal diets, possibly as the result of trauma. Furthermore, examination of large numbers of stained flat preparations of the normal rat cornea not infrequently revealed fine capillaries in the peripheral stroma. While the latter changes are not comparable to the massive vascularization of the cornea in rats with tryptophan deficiency and, in some other conditions, they signify that the rat cornea is particularly susceptible to vascularization, probably more so than the human.

Special Features of Tryptophan Deficiency Cataract.—Tryptophan deficiency cataract showed an outspoken variability among the rats of one age group and one species: It occurred in at least two morphologically different forms: an acute and a chronic. This variability points to the importance of factors other than the experimental dietary deficiency alone in the development and shaping of this cataract.

Tryptophan deficiency cataract is associated with several manifestations which have some relation to grow activity: inhibition of body growth and manifestations in epithelial tissues, which (like the crystalline lens) continue to grow throughout life (hair; testicular epithelium). This points to the (direct or indirect) importance of tryptophan in the metabolism of these growing tissues. It may be noted in this connection that some indole compounds, chemically related to tryptophan, act as growth substances in plants (heteroauxins).[22]

The presence of these features and the absence of certain characteristics of other types of cataract permit a comparison of tryptophan deficiency cataract and other experimental forms, which will be discussed in the next section.

CLASSIFICATION OF EXPERIMENTAL CATARACTS IN THE RAT

In the introduction, it was pointed out that studies of the development of cataract, as observed with the slit lamp, and of the allied manifestations elsewhere in the body may shed light on the pathogenic differentiation of experimental forms of cataract. In the present paper tryptophan deficiency cataract in the rat has been described from these points of view. It is of interest to compare these observations with those on other experimental cataracts in the same species.

In the accompanying table, the nine experimental cataracts of the rat thus far recognized have been classified on the basis of their morphologic and developmental features, as well as their allied manifestations. The three main groups of cataract in this classification are the diabetic, the tetanic and the dystrophic. Studies on cataracts of the diabetic group will be reported on later in more detail.

The table may be briefly discussed. Tryptophan deficiency cataract has in common with thallium cataract and with riboflavin deficiency cataract its outspoken variability and its association with inhibition of body growth and with manifestations in certain other organs, namely, the cornea, the skin and the testicles. The histopathologic changes in the skin associated with experimental riboflavin deficiency have been described by Sullivan and Nicholls,[23] and the observations in the testicles and other organs, by Shaw and Phillips.[24] The allied manifestations occurring with thallium poisoning have been reviewed by A. Buschke and Peiser.[25]

Although some of the allied manifestations of this group of cataracts are observed also with tetanic caatract, the latter is characterized by the chemical changes

22. Kögl, F.; Haagen-Smit, A. J., and Erxleben, H.: Ztschr. f. physiol. Chem. **228**:90, 1934.

23. Sullivan, M., and Nicholls, J.: J. Invest. Dermat. **4**:181, 1941.

24. Shaw, J. H., and Phillips, P. H.: J. Nutrition **22**:345, 1941.

25. Buschke, A., and Peiser, B.: Ergebn. d. allg. Path. u. path. Anat. **25**:1, 1931.

Experimental Cataracts Observed in the Rat *

Experimental Condition	Authors	Special Features of Cataract with Respect to Morphologic Features, Development and Occurrence	Allied Pathologic Manifestations
	Diabetic Group		
Galactose cataract	Mitchell, H. S., and Dodge, W. M.: J. Nutrition **9**: 37, 1935. Mitchell, H. S.: ibid. **12**: 447, 1936; Proc. Soc. Exper. Biol. & Med. **41**: 507, 1939. Yudkin, A. M., and Arnold, C. H.: Cataracts Produced in Albino Rats on Ration Containing High Proportion of Lactose or Galactose, Arch. Ophth. **14**: 960 (Dec.) 1935. Stillmann and Weekers.[4a] Darby and Day.[9] Sasaki.[28b] Gifford, S. R., and Bellows, J.: Histologic Changes in Lens Produced by Galactose, Arch. Ophth. **21**: 346 (Feb.) 1941.	Strong vacuolization in superficial cortex, particularly at equator of lens, in some stages (fig. 11 *C*)	Increased appetite; polydipsia; polyuria; high monosaccharide concentration in blood
Xylose cataract	Darby, W. J., and Day, P. L.: Proc. Soc. Exper. Biol. & Med. **41**: 507, 1939; footnote 9, present report.	Strong vacuolization in superficial cortex, particularly at equator of lens (fig. 11 *D*)	Polydipsia; polyuria; high monosaccharide concentration in blood
Cataract accompanying postoperative diabetes (pancreatectomy)	Richter, C. P., and Buschke, W.: To be published elsewhere.	Cataract has been observed in adult rats; strong vacuolization in superficial cortex and at equator in some stages (fig. 11 *A* and *B*)	Increased appetite; polydipsia; polyuria; high monosaccharide concentration in blood
	Tetanic Group		
Cataract associated with dietary tetany	von Bahr.[26b]	Opacities appear in anterior superficial cortex in close correlation with attacks of tetany (manifest or latent)	Low calcium-phosphate ratio in blood; neuromuscular hyperexcitability; lesions of incisor teeth
Cataract associated with postoperative tetany (parathyroidectomy)	Erdheim.[1] Goldmann.[3]	Opacities appear in anterior superficial cortex in close correlation with attacks of tetany (manifest or latent) (fig. 10)	Low calcium-phosphate ratio in blood; neuromuscular hyperexcitability; arrest of body growth; lesions of incisor teeth
	Dystrophic Group		
Cataract due to chronic thallium poisoning	Buschke, A.[2] Ginsberg and Buschke.[17] Donski, J.: Arch. f. Ophth. **128**: 294, 1932. Mamoli, L.: Ann. di ottal. e clin. oculi. **55**: 35, 1927. von Mellin, A.: Experimentelle Untersuchungen über die Entstehung des Thallium-Stares nebst Bemerkungen über eine neue vererbbare und angeborene Starform bei weissen Ratten, Munich, K. Alberrstoetter, 1926.	Cataract occurs only in growing rats; variability in occurrence outspoken; cortical and nuclear (fig. 9)	Arrest of body growth; alopecia; vascularization of cornea, keratitis and iritis; bone lesions; atrophy of gonads; endocrine lesions
Cataract associated with riboflavin deficiency	Day, P. L.; Langston, W. C., and O'Brien, C. S.: Am. J. Ophth. **14**: 1005, 1931. Day, P. L.: J. Nutrition **12**: 395, 1936. Day and Darby.[9] Bourne, M. C., and Pyke, M. A.: ibid. **29**: 1865, 1935. György, P.: ibid. **29**: 741, 1935. Cosgrove, R. W., and Day, P. L.: Am. J. Ophth. **25**: 544, 1942. Richardson, L. R., and Hogan, A. G.: Research Bull. 241, Missouri Agricultural Experiment Station, 1936; cited by Bessey and Wolbach.[10] Bessey and Wolbach.[10] Baum, H. M.; Michaelree, J. P., and Brown, E. B.: Science **95**: 24, 1942.	Cataract occurs only in growing rats; variability in occurrence and morphologic character very outspoken; cortical and nuclear	Arrest of body growth; vascularization of cornea, keratitis; alopecia and dermatitis; atrophy of testicle; nerve lesions; endocrine lesions
Cataract associated with tryptophan deficiency	Curtis, Hauge and Kraybill.[6a] Berg and Potgieter.[6b] Totter and Day.[7] Buschke, Albanese and Follis,[12a] Albanese and Buschke.[12b] Present report.	Cataract occurs only in growing rats; variability in occurrence and course of development outspoken; cortical and nuclear	Arrest of body growth; vascularization of cornea, keratitis; alopecia and other cutaneous lesions; atrophy of testicles; lesions of incisor teeth
	Unclassified		
Napthalene cataract	Goldmann.[29]	Perinuclear cataract and nuclear sclerosis	† Transitory hyperglycemia; disturbance in phosphorylative processes in lens and in metabolism of sulfhydryl compounds and ascorbic acid

* This table does not include the acute reversible opacities of the lens: "cooling cataract" (Goldschmidt [15]); cataract associated with epinephrine shock (tum Suden, C., and Wyman, L. C.: Endocrinology **27**: 628, 1940); salt cataract (Goldmann and Rabinowitz [16]) and other forms of acute osmotic opacities of the lens in the rat (Bellows, J., and Chinn, H.: Biochemistry of Lens: Production of Lens Opacities by Injection of Hypertonic Solutions, Arch. Ophth. **25**: 796 [May] 1941). Demole and Knapp (Ophthalmologica **101**: 65, 1941) have recently reported on cataracts and other ocular lesions, including corneal vascularization in some rats on vitamin E-deficient diets.

† The allied manifestations of naphthalene cataract have been described chiefly in the rabbit.

in the blood (low calcium-phosphate ratio) and by the neuromuscular hyperexcitability, characteristic of tetany. In both forms of experimental tetany the appearance of the cortical opacities of the lens is linked closely with individual attacks of tetany rather than with some chronic dystrophic condition. This is the reason for the zonular arrangements of the opacities, frequently noted in cataract associated with experimental tetany [26] (fig. 10).

On the other hand, galactose and xylose cataract and cataract associated with experimental diabetes are characterized by the high monosaccharide concentration of the blood [27] and by the associated polyuria and polydipsia.[28] In addition, these three types of cataract are characterized by the occurrence of a great number of vacuoles and clusters of vacuoles at the equator and in the superficial cortex of the lens at some stages of the development of the cataract. Figure 11 may serve

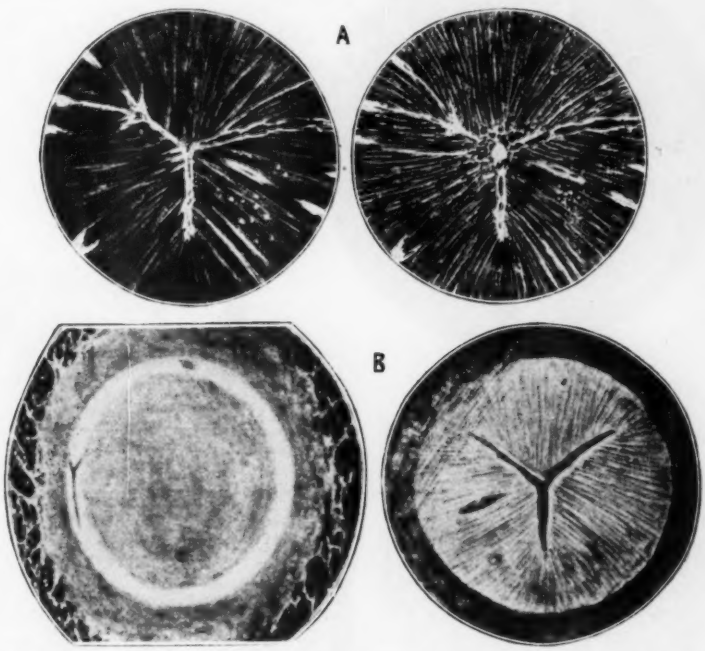

Fig. 9.—Cataract produced by chronic thallium poisoning in the rat (from Donski, J.: Arch. f. Ophth. **128**:294, 1932). *A*, early stages; *B*, later stages.

to illustrate this. There is strongly suggestive evidence that osmotic disturbances play an important role in the pathogenesis of the diabetic group of cataracts.

Allied manifestations of naphthalene cataract have been described mainly in the rabbit, though the occurrence of this cataract has also been reported in the rat.[29] The observations on early naphthalene poisoning can be summarized as follows:

26. (*a*) Goldmann,[3] (*b*) von Bahr, G.: Acta ophth., 1936, supp. 11, p. 1. (*c*) Siegrist, A.: Der graue Altersstar, Berlin, Urban & Schwarzenberg, 1928.

27. (*a*) Darby and Day.[9] (*b*) Day, P. L.: J. Nutrition **12**:395, 1936. (*c*) Richter, C. P., and Schmidt, E. C. H., Jr.: Endocrinology **28**:178, 1941.

28. (*a*) Süllmann and Weekers.[4a] (*b*) Sasaki, T.: Arch. f. Ophth. **138**:351, 365 and 380, 1938. (*c*) Bakker, A.: ibid. **140**:531, 1939. (*d*) Richter, C. P.: Am. J. Physiol. **133**: 29, 1941.

29. Goldmann, H.: Klin. Monatsbl. f. Augenh. **83**:433, 1929.

1. Disturbances in the early phases of carbohydrate metabolism. These include: transitory hyperglycemia [30]; disturbance of phosphorylative processes in the lens,[31] and increase in the lactic acid content of the lens, aqueous and blood.[31]

2. Disturbances of the redox substances of the lens: formation and excretion of mercapturic acid by the binding of sulfydryl compounds [32]; loss of sulfydryl compounds from the lens [31]; loss of ascorbic acid from the aqueous a few hours after poisoning,[33] and loss of reducing power of the lens against ascorbic acid.[34]

It is likely that these two groups of manifestations of naphthalene poisoning are related to each other. Obviously, some of these manifestations point to a

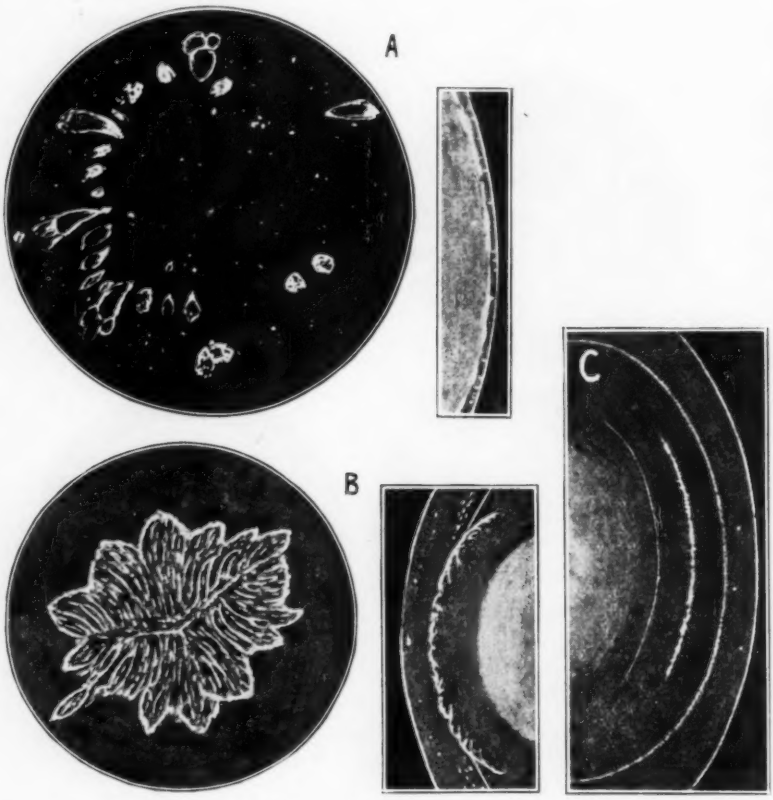

Fig. 10.—Cataract associated with experimental tetany in a parathyroidectomized rat (from Goldmann [3]). *A*, early stage; *B* and *C*, later stages.

pathogenic relation to the diabetic group of cataract; however, the appearance with the slit lamp and the absence of conspicuous polydipsia indicate that osmotic dis-

30. Michael, D., and Vancea, P.: Klin. Monatsbl. f. Augenh. **78**:285, 1927. Komura, K.: Arch. f. Ophth. **120**:766, 1928.

31. Müller, H. K.: Arch. f. Ophth. **140**:191, 1939.

32. Bourne, M. C., and Young, L.: Biochem. J. **28**:803, 1934. Stekol, J. A.: J. Biol. Chem. **110**:463, 1935; **121**:87, 1937.

33. Müller, H. K., and Buschke, W.: Arch. f. Augenh. **108**:368, 1934.

34. Müller, H. K.: Arch. f. Augenh. **109**:304, 1935.

turbances play a less important role in the production of naphthalene cataract. At present, this cataract must be left unclassified.

On the basis of the special features of the cataracts and of their allied manifestations, tryptophan deficiency cataract must be differentiated both from the diabetic and from the tetanic group of experimental cataracts in the rat. On the other hand, it can be classified with thallium cataract and with the cataract developing in the presence of riboflavin deficiency, and these cataracts may be said to constitute the dystrophic group.

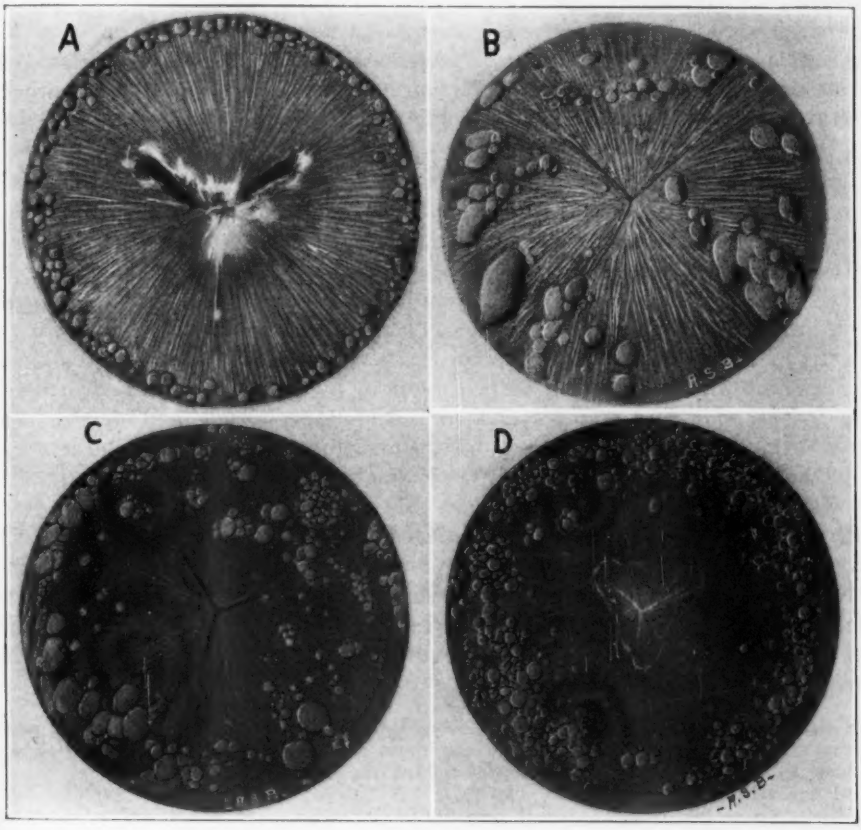

Fig. 11.—*A* and *B*, cataract associated with experimental diabetes in pancreatectomized rats (from Richter and Buschke, to be published). *C*, cataract produced in a 55 Gm. rat fed a 50 per cent galactose diet for four days. The drawing was made eight days later. *D*, cataract produced in a 38 Gm. rat fed a 50 per cent xylose diet for six days. The drawing was made five days later.

Among the symptoms of cataracts of the dystrophic group just enumerated are vascular and inflammatory reactions, which are not commonly classified as dystrophic. Similar lesions, however, are well known features of various deficiency diseases, both in men and in experimental animals. The occurrence of such symptoms, e. g., keratitis, corneal vascularization and dermatitis, as part of certain syndromes which are characterized by other, more familiar, dystrophic signs should, therefore, occasion no surprise.

THE DYSTROPHIC GROUP OF CATARACTS

The similarity of the distribution of manifestations of the dystrophic group of cataracts and the embryonic relation of some of the affected structures suggest a metabolic relation both of the affected tissues and of the various syndromes.

According to Bietti,[35] rats can be protected against cataract and other manifestations of thallium poisoning by brewers' yeast and, to a lesser degree, by liver injection N. N. R., by autoclaved brewers' yeast or by pure riboflavin. The strong protective effect of brewers' yeast against the cataract was found to be confined to thallium cataract, and practically no protection against the cataracts produced by naphthalene, galactose, lactose and alimentary hypocalcemia was observed.

In a subsequent report the human "metabolic" cataracts will be correlated with the classification of experimental cataracts suggested here, and the relation of human dystrophic cataracts to experimental dystrophic cataracts will be discussed.

SUMMARY

Cataract associated with tryptophan deficiency occurs in at least two forms, the acute and the chronic. Details of the morphologic character and development of the cataract are described. Arrest of the cataract associated with acute tryptophan deficiency by administration of tryptophan has been observed. No complete loss of bound indole occurs even in the advanced stages of tryptophan deficiency cataract.

Cataracts associated with tryptophan deficiency, riboflavin deficiency and thallium poisoning are similar with respect both to the presence of allied symptoms in the cornea, skin and testicles and to certain morphologic and developmental features, by which they are distinguished from diabetic and tetanic cataracts.

On the basis of their morphologic character and development and of the allied symptoms, a classification of the experimental cataracts in the rat is suggested. The three principal groups in this classification are the diabetic, the tetanic and the dystrophic groups. The diabetic group includes galactose and xylose cataracts and cataract associated with experimental diabetes; the tetanic group, the cataracts occurring in the course of dietary and postoperative tetany, and the dystrophic group, the cataracts accompanying riboflavin deficiency, chronic thallium poisoning and tryptophan deficiency.

The slit lamp drawings of cataracts and corneal vascularization associated with tryptophan deficiency and of the cataracts in diabetes and those produced by galactose and xylose diets were made by Mrs. Annette S. Burgess.

35. Bietti, G.: Med. sper., Arch. ital. **7**:3, 1941; abstracted, Zentralbl. f. d. ges. Ophth. **46**: 502, 1941.

DYSTROPHIC CATARACTS AND THEIR RELATION TO OTHER "METABOLIC" CATARACTS

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In the various papers on cataract, generalizations concerning cataract as the result of local or general nutritional disturbances, endocrine disturbances, osmotic disturbances and others have led to much confusion. In a previous paper [1] I outlined a classification of experimental cataracts as diabetic, tetanic and dystrophic, on the basis of their allied manifestations and developmental features. The object of this paper is to correlate this classification with the "metabolic" cataracts observed in human beings and, in particular, to discuss the relation of the experimental and the clinical cataracts of the dystrophic group. The object of this classification is not primarily a diagnostic one, but is the unification and differentiation of features which should help in delimiting the various groups of cataract and the consideration of these groups from the standpoint of the difference in pathogenesis.

In the bibliographic analysis [2] that follows, attention has been especially focused on the dystrophic group. The diabetic and the tetanic group are presented briefly chiefly for purposes of contrast.

BIBLIOGRAPHIC ANALYSIS OF TYPES OF CATARACT

There are some forms of cataract, e. g., those associated with dinitrophenol and with ergot poisoning, which in the present state of knowledge show no relation whatever to any one of the three groups which have been differentiated in this discussion. Of the unclassified forms, only the cataract due to damage from roentgen rays is discussed; this form is described briefly to illustrate the possibility that further knowledge may establish a relation between damage produced by roentgen rays and the dystrophies.

I. The Diabetic Group.

1. True diabetic cataract in man.[3]
2. Experimental diabetic cataract in dogs [4] and rats.[5]

From the Wilmer Ophthalmological Institute of the Johns Hopkins University School of Medicine.

1. Buschke, W.: Classification of Experimental Cataracts in the Rat: Recent Observations of Cataract Associated with Tryptophan Deficiency and with Some Other Experimental Conditions, Arch. Ophth. **30**:735 (Dec.) 1943.

2. No attempt is made here to give a complete bibliography of the literature on "metabolic" cataracts and on theories of cataract formation. The reader may be referred to reviews and discussions by the following authors: (*a*) Siegrist, A.: Der graue Altersstar, Berlin, Urban & Schwarzenberg, 1928. (*b*) Bourne, M. C.: Physiol. Rev. **17**:1, 1937. (*c*) Bellows, J. G., and Chinn, H.: Theories of Cataract, Arch. Ophth. **26**:1066 (Dec.) 1941. (*d*) Duke-Elder, W. S.: Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1941, vol. 3.

3. Schnyder, F. W.: Klin. Monatsbl. f. Augenh. **70**:45, 1923. O'Brien, C. S.; Molsberry, J. M., and Allen, J. H.: Diabetic Cataract: Incidence and Morphology in One Hundred and Twenty-Six Young Diabetic Patients, J. A. M. A. **103**:892 (Sept. 22) 1934.

4. Chaikoff, L., and Lachman, G. S.: Proc. Soc. Exper. Biol. & Med. **31**:237, 1933.

5. Richter, C. P., and Buschke, W.: To be published.

3. Lactose and galactose cataract in rats,[6] rabbits [7] and mice.[7]
4. Xylose cataract in rats.[8]

The most conspicuous allied manifestations [1] are increased appetite, polydipsia and polyuria and a high monosaccharide concentration in the blood. A characteristic morphologic feature of the cataracts due to galactose and xylose and to experimental diabetes in the rat is the accumulation in clusters of a large number of cortical and subcapsular vacuoles, particularly at the equator of the lens, in some stages.[1] It is suggestive that the subcapsular vacuoles which are seen in large numbers in some cases of human diabetic cataract, and which have been stated to be characteristic by some authors,[9] are the equivalent of the much more massive vacuolar changes in the experimental cataract forms of this group.

The evidence points strongly to osmotic disturbances as a pathogenic factor in this entire group of cataracts. This possibility has been noted before in connection with individual members of the group.[10]

II. The Tetanic Group.

1. Cataract associated with postoperative hypoparathyroidism in man.[11]
2. Cataract associated with idiopathic tetany in man.[11]
3. Cataract associated with experimental postoperative hypoparathyroidism in dogs,[12] rats [13] and rabbits.[13b]
4. Experimental dietary tetanic cataract in rats [11a] and rabbits.[14]
5. Possibly some zonular cataracts.[15]

It is possible, but so far undecided, that the cataracts which have been observed in cases of Gee-Herter disease (Herter-Heubner disease, intestinal infantilism, celiac disease, nontropical sprue) and which are accompanied by tetany belong to the tetanic group.[16] The same may be said of cataract associated with osteomalacia.[17]

The most conspicuous allied manifestations are neuromuscular hyperexcitability and a lowered ratio of calcium to phosphate in the blood. This group of cataracts has some allied disturbances (lesions of the skin, hair, nails and teeth) in common with cataracts of the dystrophic group.

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6. Mitchell, H. S., and Dodge, W. M.: J. Nutrition **9**:37, 1935. Yudkin, A. M., and Arnold, C. H.: Cataracts Produced in Albino Rats on a Ration Containing a High Proportion of Lactose or Galactose, Arch. Ophth. **14**:960 (Dec.) 1935.

7. Unpublished observations.

8. Darby, W. J., and Day, P. L.: Proc. Soc. Exper. Biol. & Med. **41**:507, 1939; J. Biol. Chem. **133**:503, 1940.

9. (*a*) Meesmann, A.: Die Mikroskopie des lebenden Auges an der Gullstrandschen Spaltlampe, Berlin, Urban & Schwarzenberg, 1927, p. 123. (*b*) Duke-Elder, W. S.: Recent Advances in Ophthalmology, ed. 3, Philadelphia, P. Blakiston's Son & Co., 1934, p. 336; footnote *2 d*, p. 3206.

10. Süllmann, H., and Weekers, R.: Ztschr. f. Augenh. **95**:58, 1938. Bakker, A.: Arch. f. Ophth. **140**:531, 1939. Bellows and Chinn. [2c] Duke-Elder,[9b] p. 354. Duke-Elder,[2d] p. 3208.

11. Reviewed by: (*a*) von Bahr, G.: Acta ophth., 1936, supp. 11, p. 1. (*b*) Meesmann, A.: Hypocalcaemie und Linse, Stuttgart, Ferdinand Enke, 1938. (*c*) Duke-Elder.[2d]

12. Siegrist.[2a] Duke-Elder.[2d]

13. (*a*) Erdheim, J.: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **16**:632, 1906. (*b*) Goldmann, H.: Arch. f. Ophth. **122**:146, 1929.

14. Swan, K. C., and Salit, P. W.: Am. J. Ophth. **24**:611, 1941.

15. von Bahr.[11a] Meesmann.[11b]

16. Bennett, T. I.; Hunter, D., and Vaughan, J. M.: Quart J. Med. **1**:603, 1932. Bangerter, A.: Ophthalmologica **98**:291, 1940.

17. Maxwell, J. P., and Pi, H. T.: Proc. Roy. Soc. Med. **33**:777, 1940.

A morphologic and developmental feature of the tetanic group is the zonular development of the opacities in connection with the periodic action of the damaging factor or factors in the experimental cataracts.[18] and probably in some clinical forms,[11] and the close time relation between the development of the opacities of the lens and the occurrence of the systemic changes typical of tetany.

It is of interest in this connection that cyclic changes in activity, mood and craving for calcium associated with both experimental and clinical tetany have been observed.[19]

III. The Dystrophic Group.

A. Experimental conditions.

1. Cataract associated with experimental riboflavin deficiency in rats.[20]
2. Cataract due to chronic thallium poisoning in rats and mice.[21]
3. Cataract associated with experimental tryptophan deficiency in rats.[1]

B. Clinical cataract syndromes with predominantly atrophic dermatoses.

4. Cataracta syndermatotica associated with poikiloderma vasculare atrophicans (Rothmund syndrome).[22]
5. Cataracta syndermatotica associated with scleroderma (Werner syndrome).[23]

C. Clinical cataract syndromes with atopic dermatitis.

6. Cataracta syndermatotica with atopic eczema, neurodermatitis, chronic neurodermatitis (Andogsky syndrome)[24] and Besnier's prurigo with ichthyosis.[25]

D. Clinical cataract syndromes associated with other cutaneous conditions.

7. Cataracta syndermatotica in rare cases of keratosis follicularis [26] (Darier's disease), telangiectasis [27] and myxedema.[28]
8. Cataract with developmental anomalies of the hair, such as curly hair and aplasia pilaris.[29]

E. Other clinical cataract syndromes of the dystrophic group.

9. Cataract associated with mongolism.[30]
10. Cataract associated with dystrophia myotonica.[31]

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18. Siegrist.[2a] Duke-Elder.[2d] Goldmann.[13b]
19. Richter, C. P.; Honeyman, W. M., and Hunter, H.: J. Neurol. & Psychiat. **3**:19, 1940.
20. Reviewed by Cosgrove, R. W., and Day, P. L.: Am. J. Ophth. **25**:544, 1942. Cataract has recently been observed also in pigs on riboflavin-deficient diets (Wintrobe, M. M.; Buschke, W.; Follis, R. H., Jr., and Humphreys, S.: To be published.
21. Reviewed by: (*a*) Buschke, A., and Peiser, B.: Ergebn. d. allg. Path. u. path. Anat. **25**:1, 1931. (*b*) Donski, J.: Arch. f. Ophth. **128**:294, 1932.
22. Reviewed by Schnyder, F. W.: Schweiz. med. Wchnschr. **16**:719, 1935.
23. Reviewed by: Siegrist.[2a] Oppenheimer, B. S., and Kugel, W. H.: Am. J. M. Sc. **202**: 629, 1941. Agatston, S. A., and Gartner, S.: Precocious Cataracts and Scleroderma (Rothmund's Syndrome; Werner's Syndrome): Report of Case, Arch. Ophth. **24**:492 (March) 1939.
24. Reviewed by Beetham, W. B.: Atopic Cataracts, Arch. Ophth. **24**:21 (July) 1940.
25. Kugelberg, I.: Klin. Monatsbl. f. Augenh. **92**:484, 1934.
26. Gjersing, O. M., cited by Franceschetti, A., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 1, p. 754.
27. Nieden, A.: Zentralbl. f. Augenh. **11**:353, 1887. Friedenwald, J. S.: Unpublished data.
28. Löwenstein, A.: Klin. Monatsbl. f. Augenh. **76**:539, 1926.
29. Werner, S.: Acta ophth. **6**:382, 1928. Papastratigakis, C.: Paris méd. **12**:475, 1922. Cederkreutz, A.: Zentralbl. f. Haut- u. Geschlechtskr. **21**:841, 1927.
30. Reviewed by van der Scheer, W. M.: Klin. Monatsbl. f. Augenh. **62**:155, 1919.
31. Reviewed by Allen, J. H., and Barer, C. G.: Cataract of Dystrophia Myotonica, Arch. Ophth. **24**:867 (Nov.) 1940.

CATARACTS OF THE DYSTROPHIC GROUP

A brief discussion of the various cataracts of the dystrophic group follows, with some bibliographic references.

A. EXPERIMENTAL CONDITIONS

1. Riboflavin Deficiency.—Cataract: Day and associates [32] observed cataract in animals fed a diet deficient in the heat-stable fraction of the vitamin B complex, then called vitamin G. The incidence of this form of cataract varied from zero to almost 100 per cent in various laboratories.[33] The possible role of the susceptibility of special strains has been pointed out.[33d]

The specific relation of the cataract to riboflavin has been demonstrated by the protective and curative (arresting) effect of this substance.[34] However, the relation of this cataract to riboflavin deficiency is not simple; while some authors have claimed that the small amount of riboflavin present in certain caseins may explain failure to produce cataract,[35] others have asserted that the incidence of cataract is higher if traces of riboflavin are added to diet.[36]

It appears that a factor or factors other than riboflavin may influence the manifestation of this cataract. With this qualification, one may speak of a riboflavin deficiency cataract.

According to O'Brien,[32b] the development of the cataract does not follow any rule: Nuclear, cortical and anterior and posterior subcapsular opacities were observed in various combinations in different animals, and even in the two eyes of the same animal.

Other manifestations: Signs of riboflavin deficiency in the organs are: (1) arrest of body growth [37]; (2) dermatosis with alopecia [38]; (3) keratitis [20]; (4) lesions of the testicles [39]; (5) nerve lesions,[39] and (6) lesions in the endocrine organs, such as the thymus.[39]

2. Experimental Chronic Thallium Poisoning.—Cataract: Cataract in rats and mice with chronic thallium poisoning was observed by A. Buschke.[40]

The cataract begins with anterior subcapsular changes. The cortex becomes progressively opaque; suddenly perinuclear and fine nuclear clouding occurs.[21b]

Thallium cataract did not develop in older rats. Some investigators were not able to produce mature thallium cataract,[41] and still others observed great individual

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32. (*a*) Day, P. L.; Langston, W. C., and O'Brien, C. S.: Am. J. Ophth. **14**:1005, 1931. (*b*) O'Brien, C. S.: Experimental Cataract in Vitamin G Deficiency, Arch. Ophth. **8**:880 (Dec.) 1932. (*c*) Day, P. L.: J. Nutrition **12**:395, 1936. (*d*) Day, P. L., and Langston, W. C.: ibid. **7**:97, 1934.

33. (*a*) Cosgrove and Day.[20] (*b*) György, P.: Biochem. J. **29**:741, 1935. (*c*) Bourne, M. C., and Pyke, M. A.: ibid. **29**:1865, 1935. (*d*) Bessey, O. A., and Wolbach, S. B.: J. Exper. Med. **69**:1, 1939. (*e*) Richardson, L. R., and Hogan, A. C., cited by Bessey and Wolbach. (*f*) Eckardt, R. E., and Johnson, L. V.: Nutritional Cataract and Relation of Galactose to Appearance of Senile Suture Line in Rats, Arch. Ophth. **21**:315 (Feb.) 1939. (*g*) Footnote 32.

34. Day, P. L.; Darby, W. J., and Cosgrove, K. W.: J. Nutrition **15**:83, 1938.

35. Day, P. L., and Darby, W. J.: Biochem J. **32**:1171, 1938.

36. Baum, H. M.; Michaelree, J. F., and Brown, E. B.: Science **95**:24, 1942.

37. Kuhn, R.; Rudy, H., and Wagner-von Jauregg, T.: Ber. d. deutsch. chem. Gesellsch. **66**:1950, 1933.

38. Sullivan, M., and Nicholls, J.: J. Invest. Dermat. **4**:181, 1941.

39. Shaw, J. H., and Phillips, P. H.: J. Nutrition **22**:345, 1941.

40. Buschke, A.: Arch. f. Dermat. u. Syph. **116**:477, 1913.

41. von Mellin, A.: Experimentelle Untersuchungen über die Entstehung des Thallium-Stares nebst Bemerkungen über eine neue vererbbare und angeborene Starform bei weissen Ratten, Munich, K. Alberrstoetter, 1926.

differences in the development of the cataract and familial variation in susceptibility [42]; in particular, the rapidity with which the opacities developed was subject to familial variation.

Other Manifestations: Changes in other organs are: (1) arrest of body growth [21a]; (2) alopecia [21a]; (3) keratitis and corneal vascularization [42a]; (4) iritis [42a]; (5) testicular lesions [21a]; (6) lesions in other endocrine organs,[21a] including the adrenals, the thyroid, the pancreas, the islets of Langerhans and the parathyroids; (7) nerve lesions [21a]; (8) rachitic changes in bone,[21a] and (9) inflammatory and metaplastic changes in the gastrointestinal tract.[21a]

3. Experimental Tryptophan Deficiency.—Cataract: [43] The morphologic features and development of this cataract have been described in a previous report.[1] An acute and a chronic type of cataract have been observed. The acute type involves first the cortex and later the perinuclear and nuclear zones. The chronic type is confined to the cortex.

Allied Manifestations: Changes in other organs are: (1) arrest of body growth [44]; (2) corneal vascularization and keratitis [43]; (3) cutaneous lesions and alopecia,[44] and (4) testicular atrophy and aspermatogenesis.[45] Lesions in the incisor teeth have been observed, too, in animals on a tryptophan-deficient diet [46]; the relation of these lesions to tryptophan deficiency demands further study.

The occurrence of a hypochromic anemia in animals on tryptophan-deficient diets has been reported.[46a]

Histopathologic studies which are being carried on by Dr. L. Emmett Holt Jr. and his associates may reveal other pathologic manifestations of tryptophan deficiency, in addition to those mentioned here.

B. CLINICAL CATARACT SYNDROMES WITH PREDOMINANTLY ATROPHIC DERMATOSES

4. Rothmund Syndrome.[47]—Cataract: The cataract develops rapidly in early childhood (between the second and the sixth year of age) and becomes mature within a few days to months. It begins in the anterior and the posterior cortex (Siegrist [2a]). Later the nucleus becomes milky, and a dense perinuclear, shell-like opacity is formed.

Other Manifestations: Changes in other organs are: (1) dermatosis: poikiloderma vasculare atrophicans (Jacobi) and (3) atrophy and underdevelopment of the gonads in both sexes.[22]

The disease is probably inherited as a recessive.[48]

5. Werner Syndrome.[49]—Cataract: The cataract develops in the third or fourth decade of life. Opacities of various sizes are seen in the posterior and the anterior

42. (*a*) Ginsberg, S., and Buschke, A.: Klin. Monatsbl. f. Augenh. **71**:385, 1923. (*b*) Mamoli, L.: Ann. di ottal. e clin. ocul. **55**:35, 1927.

43. (*a*) Totter, J. R., and Day, P. L.: J. Biol. Chem. **140**:cxxxiv, 1941; (*b*) J. Nutrition **24**:159, 1942. (*c*) Buschke, W.; Albanese, A. A., and Follis, R. H., Jr.: Federation Proc. **1**:175, 1942. (*d*) Albanese, A. A., and Buschke, W.: Science **95**:584, 1942.

44. Berg, C. P.; Rose, W. C., and Marvel, C. S.: J. Biol. Chem. **85**:207 and 219, 1929.

45. Shettles, L. B.: Proc. Ann. Conf. Biol. Spermatozoa, 1942, p. 28.

46. Buschke and associates.[43c]

46a. Albanese, A. A.; Randall, R. McI., and Holt, L. E., Jr.: Science **97**:312, 1943.

47. Rothmund, A.: Arch. f. Ophth. (pt. 1) **14**:159, 1868. Seefelder, R.: Ztschr. f. Augenh. **86**:81, 1935.

48. Vogt, A.: Lehrbuch und Atlas d. Spaltlampenmikroskopie des lebenden Auges, Berlin, Julius Springer, 1931, vol. 2.

49. Werner, C. W. O.: Ueber Katarakt in Verbindung mit Sklerodermie, Inaug. Dissert., Kiel, Schmidt & Klaunig, 1904.

cortex; larger opacities, in the form of "bread crumbs," are seen at the posterior pole (Kugelberg [25]). Nuclear cataract has been described, in addition to cortical opacities.[50]

Occurrence of the disease in siblings and in several generations of the same family has been reported.[51]

Other Manifestations: Changes in other organs are: 1. Dermatosis: scleroderma [52]; changes in the hair, such as premature graying and baldness,[52] and ulcers.[51] 2. Atrophy of the testicles and aspermatogenesis; premature sexual involution in the female.[52] 3. Endocrine manifestations [51]: hyperparathyroidism; adenomas of thyroid and adrenal cortex; abnormal blood sugar curve. 4. Keratitis bullosa.[53]

Marchionini and Lux [54] described a case of scleropoikiloderma (Arndt-Jaffé) with anterior and posterior cortical and subcapsular cataract in a patient aged 30. Clinically and histologically the skin exhibited features of scleroderma and poikiloderma, and changes in the endocrine glands similar to those associated with scleroderma were noted.

Possibly the few instances of cataract in cases of telangiectasis of the skin of the face, hands and arms (Nieden; Friedenwald [27]) also belong in this group.

C. CATARACT SYNDROMES WITH DERMATOSES OF THE ATOPIC DERMATITIS TYPE

6. *Andogsky Syndrome*.[55]—Cataract: Two types of cataract have been described (Beetham [56a]). The one starts at the posterior pole, spreading later to the anterior cortex and the nucleus; the other begins as an anterior subcapsular rosette, in which a dense, umbrella-shaped opacity with concave margins develops. Later the anterior and the posterior cortex become opaque, and the cataract matures within several months or years. It occurs in the second or third decade of life.

Other Manifestations: Changes in other organs are: 1. Superficial keratitis [57] (keratitis epithelialis vesiculosa disseminata; keratitis superficialis punctata). It appears concomitantly with the attacks of eczema. Subjective symptoms may be insignificant, and the corneal changes may escape notice without the use of the slit lamp. 2. Dermatosis [58]: atopic dermatitis (synonyms: atopic eczema; lichen chronicus [Vidal]; *prurigo diathésique* [Besnier]; *neurodermite diffuse* [Brocq-Jacquet]; atopy [Coca]; allergic eczema on a constitutional basis [Juelg]).

No unequivocal evidence for the presence of endocrine disorders could be found in the literature, although claims to that effect have been made.[28]

3. Manifestations of hypersensitiveness, either in the patients themselves or in their family—(asthma, hay fever, eosinophilia, multiple cutaneous reactions).[59]

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50. Vossius, A.: Ztschr. f. Augenh. **43**:640, 1920.

51. (*a*) Footnote 23. (*b*) Mamou, H.: Sclerodermie et cataracte, Thesis, Paris, 1931. (*c*) Krebs, E.; Hartmann, E., and Thiebaut, F.: Rev. neurol. **53**:606, 1930.

52. Werner.[49] Footnote 51.

53. Kleiber: Klin. Monatsbl. f. Augenh. **65**:923, 1920.

54. Marchionini, A., and Lux, L.: Arch. f. Dermat. u. Syph. **176**:309, 1937.

55. Andogsky, N.: Klin. Monatsbl. f. Augenh. **52**:842, 1914.

56. (*a*) Beetham.[24] (*b*) Kugelberg.[25] (*c*) Vogt, A.: Schweiz. med. Wchnschr. **51**:669, 1921. (*d*) Vogt,[48] p. 557.

57. Vogt,[48] 1930, vol. 1, p. 170. Pillat, A.: Wien. klin. Wchnschr. **50**:768, 1937.

58. (*a*) Coca, A. F.: Specific Diagnosis and Treatment of Allergic Diseases of Skin, J. A. M. A. **103**:1275 (Oct. 27) 1934. (*b*) Footnote 56.

59. Beetham.[24] Gugelberg.[25]

No evidence for an allergic origin of the cataract could so far be adduced.[60] There now exists general agreement as to the familial character of the atopic dermatitis.[61]

Kugelberg [25] described cataract in association with ichthyosis vulgaris, Besnier's prurigo, hypogenitalism and developmental lesions of the teeth. The cataract appeared in the form of irregularly disseminated fine opacities, both in the anterior and in the posterior cortex, predominantly subcapsular. The cutaneous lesions occurred in several members of the family. Whether the combination of manifestations described by Kugelberg is due to coincidence or to a combination of two hereditary syndromes may be left open to question.

D. CLINICAL CATARACT SYNDROMES ASSOCIATED WITH OTHER CUTANEOUS CONDITIONS

7. Cataract Associated with Keratosis Follicularis (Darier's Disease [26]), Telangiectasis [27] and Myxedema.[62]—The association of cataract with myxedema is commonly referred to in papers on metabolic or dystrophic cataract. I found only 5 cases in the literature. In 3 of these 5 cases other causes (tetany; senile cataract) were not ruled out. In view of the relative frequency of myxedema and cretinism, one would expect a more frequent occurrence of cataract in either of these conditions if thyroid insufficiency were a primary factor in the development of cataract.

8 Cataract Associated with Anomalies of the Hair.[29]—A few scattered observations have been made on the association of anomalies of the hair (curly hair; alopecia, and aplasia pilaris) with juvenile cataract.

E. OTHER CLINICAL CATARACT SYNDROMES OF THE DYSTROPHIC GROUP

9. Cataract Associated with Mongolism.[63]—The most frequent form of cataract associated with mongolism [30] is the "cataracta punctata disseminata," or "snowflake cataract." A great number of dotlike or flakelike opacities appear either throughout all the layers or only in the cortex. In addition, a star of intensely white opacities appears near the anterior or the posterior pole of the lens. In a series of 60 patients with mongolism, van der Scheer [30] found cataract in none under 8 years of age, in 61 per cent of the patients from 8 to 17 years of age and in 100 per cent of the patients beyond 17 years of age. This indicates that persons with mongolism are liable to have cataract if they live long enough, i. e., beyond the age of 17 years. However, such persons die at an average age of 14 years, and 40.5 per cent die

60. Brunsting, L. A.: Atopic Dermatitis (Disseminated Neurodermatitis) of Young Adults: Analysis of Precipitating Factors in One Hundred and One Cases and Report of Ten Cases with Associated Juvenile Cataract, Arch. Dermat. & Syph. **34**:935 (Dec.) 1936. Daniel, R. K.: Allergy and Cataracts, Tr. Sect. Ophth., A. M. A., 1935, p. 50. Woods, A. C., in discussion on Daniel, p. 55. Beetham.[24]

61. (*a*) In order to avoid misunderstandings, I refer here to the classification of dermatoses associated with hypersensitivity by Coca and Sulzberger [58a]: (1) atopic dermatoses, of familial occurrence; (2) contact dermatitis, nonfamilial, and (3) fungous dermatitis, or eczematous dermatophytosis. It is to be understood that the cataract syndrome under discussion has to do with the first of these three conditions. (*b*) Holt, L. E., Jr., and McIntosh, R.: Diseases of Infancy and Childhood, ed. 11, New York, D. Appleton-Century Company, Inc., 1939.

62. (*a*) Löwenstein.[28]. (*b*) Callen, P. A.: Tr. Am. Ophth. Soc. **7**:391, 1895. (*c*) Dutoit, A.: Ztschr. f. Augenh. **32**:139, 1914. (*d*) Cantonnet, M. A.: Ann. d'ocul. **143**:475, 1920. (*e*) Nitzulescu, J.: Zentralbl. f. d. ges. Ophth. **15**:847, 1926.

63. Pearce, F. H.; Rankin, R., and Ormond, A. W.: Brit. M. J. **2**:186, 1910. Ormond, A. W.: Tr. Ophth. Soc. U. Kingdom **32**:69, 1911

even before the age of 5 years.[64] The more extensive opacities are usually observed in patients beyond the age of 12 years.

Other Manifestations[64]: Changes involving other organs are: (1) mongoloid habitus, such as peculiarities of lid fissures and epicanthus; (2) idiocy; (3) skeletal changes; (4) developmental anomalies of the teeth and retardation and irregularities of the first and the second dentition; (5) various anomalies of the skin, trophic disturbances of the nails and sparse hair and a tendency to eczema; (6) atrophy of the testicles and cryptorchism, sexual infantilism in both sexes and sterility; (7)

Synoptic Table of

Condition	Special Features of Cataract with Regard to Occurrence and Morphologic Character	Allied			
		Cornea	Skin and Adnexa	Gonads	Teeth
Experimental riboflavin deficiency	Cortical and nuclear; outspoken variability in occurrence and morphologic character; occurs only in young rats	Vascularization; keratitis	Dermatitis and alopecia	Atrophy of testicles; aspermatogenesis; anestrus	.....
Experimental thallium poisoning	Cataract begins in cortex; outspoken variability in occurrence; occurs only in young rats	Vascularization	Alopecia	Aspermatogenesis; anestrus	.....
Experimental tryptophan deficiency	Cataract begins in cortex; outspoken variability in morphologic character and development; occurs only in young rats	Vascularization; keratitis	Alopecia and other cutaneous lesions	Atrophy of testicles; aspermatogenesis	Lesions in incisor teeth*
Rothmund syndrome	Cataract begins in anterior and posterior cortex; develops in a few months; occurs in early childhood	.....	Poikiloderma vasculare atrophicans (Jacobi); atrophic nails and hair	Atrophy of testicles; aplasia of ovaries	.....
Werner syndrome	Cataract begins in anterior and posterior cortex; occurs in adult life (3d to 4th decade)	Bullous keratitis	Scleroderma; cutaneous ulcers; premature graying of hair; baldness	Atrophy of testicles	.....
Andogsky syndrome	Type 1 begins at posterior pole; type 2 begins as a stellate opacity in anterior cortex; develops within several months; occurs in adolescent or adult life	Keratitis superficialis epithelialis punctata	Atopic dermatitis	.....	†
Dystrophia myotonica (Batten-Gibb-Steinert)	Cataract begins in posterior and anterior superficial cortex; many iridescent and white dots; occurs from 3d decade on	Bullous keratitis	Premature baldness	Atrophy of testicles; loss of libido in both sexes	.....
Mongolism	Cortical and nuclear; occurs as star-shaped polar opacities and "snowflake" cataract (cataracta punctata disseminata); in general appears after age of 8 years	.....	Sparse hair; trophic anomalies of nails; tendency to eczema	Sexual underdevelopment; cryptorchism; testicular atrophy	Late and irregular dentitions

* The relation of these lesions to tryptophan deficiency demands further study.
† Developmental lesions of the teeth have been described occasionally, but the question of coincidence cannot yet be answered with certainty.

developmental lesions of the heart; (8) hypoplastic changes in the thyroid, and (9) various developmental anomalies.

Occasional manifestations of endocrine disturbance in cases of mongolism cannot be of etiologic importance, as Fanconi[65] pointed out. However, evidence points strongly either to a disturbance in the plasma of the ovum (Rosanoff[66]) or to a chromosomal mutation.[65]

64. Brousseau, K., and Brainerd, H. G.: Mongolism: A Study of the Physical and Mental Characteristics of Mongolian Imbeciles, Baltimore, Williams & Wilkins Company, 1928.
65. Fanconi, G.: Schweiz. med. Wchnschr. **69**:995, 1939.
66. Rosanoff, A. J.: Manual of Psychiatry and Mental Hygiene, New York, John Wiley & Sons, Inc., 1938, chap. 9.

10. Cataract Associated with Dystrophia Myotonica (Myotonia Atrophica) (Steinert-Batten-Gibb).[67]—Cataract: Cataract in this disease was first observed by Greenfield.[68] The cataract of myotonia becomes manifest at any time after the third decade of life. Often senile and presenile cataract is present in the ancestors. The cataract develops at a progressively earlier age in successive generations (anticipation). It begins usually as a rosette-like opacity in the posterior cortex.[48] Then innumerable dustlike, white and iridescent opacities appear both in the anterior and in the posterior cortex, leaving free the directly subcapsular zone.

Dystrophic Cataracts

Manifestations					Other Manifestations (Growth, Allergy)	Intrinsic, or Genotypical Character
Bones	Neuromuscular System	Central Nervous System	Hematopoietic System	Endocrine Organs		
.....	Degenerative lesions in peripheral nerves	.....	.....	Histologic changes in thymus	Arrest of body growth	.....
Ricket-like lesions	.....	.....	Various changes in blood picture	Histologic changes in thyroids, parathyroids, adrenals, islets of Langerhans and thymus	Iritis; arrest of body growth; metaplastic changes in prestomach	.....
.....	.....	.....	Anemia	.....	Arrest of body growth	.....
.....	.....	.....	.....	.....	.....	Hereditary (recessive)
.....	.....	.....	.....	Hyperparathyroidism; irregularities in regulation of blood sugar	.....	Probably hereditary; occurs in siblings (possibly recessive)
.....	.....	.....	.....	.....	Allergic manifestations (asthma; hay fever)	Outspoken familial occurrence; occurs in siblings and in several generations
.....	Myotonic reaction; myotonia atrophica	Changes of emotional character	.....	Changes in thyroid, inconstant	.....	Hereditary (dominant)
Various skeletal anomalies	.....	Idiocy	.....	Occasionally hypoplastic changes in thyroid	Mongoloid habitus; various developmental anomalies (heart and other organs)	Occurs in both of identical twins, but in only one of fraternal twins ‡

‡ While this does not necessarily indicate a hereditary origin, it is strong evidence of the intrinsic character of the syndrome; it is compatible with the assumption of idioplastic damage to the ovum (Rosanoff [66]) or of a chromosomal mutation (Fanconi [65]).

Other Manifestations [69]: Changes in other organs are: (1) testicular atrophy and loss of libido in both sexes [70]; (2) premature baldness [71]; (3) lesions of endocrine organs, such as the thyroid [71]; (4) myotonic atrophy with characteristic

67. Steinert, H.: Deutsche Ztschr. f. Nervenh. **37**:58, 1909. Batten, F. R., and Gibb, H. P.: Brain **32**:187, 1909.

68. Greenfield, J. G.: Rev. Neurol. & Psychiat. **9**:169, 1911.

69. Adie, W. J., and Greenfield, J. G.: Brain **46**:73, 1923.

70. (*a*) Fleischer, B.: Arch. f. Ophth. **96**:91, 1918. (*b*) Hauptmann, A.: Klin. Monatsbl. f. Augenh. **60**:576, 1918. (*c*) Adie and Greenfield.[69]

71. Fleischer.[70a] Hauptmann.[70b]

myotonic reaction, most frequent in the muscles of the thumb and in the interosseus muscles of the hand,[72] and (5) bullous keratitis.[73]

According to Vogt,[48] the muscular manifestations are not *conditio sine qua non* for the syndrome, and the changes in the lens seem to be much more frequent than the muscular alterations. In the same pedigree, cases of cataract can be found with and without muscular changes.

In spite of the presence of endocrine disturbances, no conclusive evidence exists that endocrine disturbances are the cause of the other manifestations. But it has been proved beyond doubt that the condition is hereditary.[74]

UNCLASSIFIED CATARACTS

Of the many forms of cataract which in the present state of knowledge do not appear to fit clearly into any one of the three groups previously outlined, only one is described here.

Cataract Due to Damage from Roentgen Rays.—This cataract begins at the posterior pole as a disklike, subcapsular, foamy opacity, which is prominent on its anterior surface. Fine subcapsular opacities appear also in the anterior cortex. The posterior opacity may expand over the whole posterior subcapsular zone, thus forming a shell. The cataract progresses slowly.[75]

The first visible evidence of damage to the lens due to roentgen radiation is the formation of new, abnormal suture lines as a consequence of the arrest of growth (extension in length in this case) of the affected lens fibers.[76]

The other manifestations are evident from the order of decreasing radiosensitivity of the different tissues of the body (Desjardins[77]). After the lymphoid cells and polymorphonuclear leukocytes, epithelial cells, particularly the basal epithelium of the testes, the follicular epithelium of the ovary and the basal epithelium of the skin and mucous membranes, are among the most radiosensitive tissues. The corneal changes (keratitis; epithelial changes; interstitial keratitis, and vascularization of the cornea) due to damage from roentgen radiation have been reviewed by Rohrschneider and others.[78]

A synopsis of the more important cataract syndromes of the dystrophic group is presented in the accompanying table.

COMMENT

Knowledge of the pathogenesis of the dystrophic cataracts is more incomplete than that of any of the other groups of "metabolic" cataracts: the diabetic and the tetanic.

Role of Endocrine Glands in "Metabolic" Cataracts.—The primary importance of endocrine glands in the pathogenesis of human diabetic and of some tetanic cataracts is obvious. Lesions of the endocrine glands are often encountered also

72. Vogt.[48] Footnote 70.

73. Birnbacher, T.: Ztschr. f. Augenh. **62**:44, 1927. Maillard: Klin. Monatsbl. f. Augenh. **77**:217, 1926.

74. Frey, H. C.: Arch. f. Rassen- u. Gesellsch.-Biol. **17**:1, 1925. Waardenburg, P. J.: Das menschliche Auge und seine Erbanlagen, Haag, Martinus Nijhoff, 1932.

75. Rohrschneider, W.: Klin. Monatsbl. f. Augenh. **81**:254, 1928. Meesmann, A.: ibid. **81**:259, 1928.

76. Goldmann, H., and Liechti, A.: Arch. f. Ophth. **138**:722, 1938.

77. Desjardins, A. U., cited by Scott, C. M.: Some Quantitative Aspects of the Biological Action of X and γ Rays, Medical Research Council, Special Report Series, no. 223, London, His Majesty's Stationery Office, 1937.

78. Birch-Hirschfeld, A.: Klin. Monatsbl. f. Augenh. **46**:129, 1908. Rohrschneider, W.: Arch. f. Ophth. **121**:537, 1929.

in cases of syndromes of the dystrophic group (table, column 11), and have been said to be responsible for the cataracts. However, the cataracts and the other manifestations of the dystrophic syndromes cannot be arrested or remedied by administration of any known endocrine glandular extract. Furthermore, cataract thus far has not been produced experimentally by the complete removal of any endocrine gland other than the pancreas or the parathyroids. An explanation of the syndromes of dystrophic cataract on a primarily endocrine basis is, therefore, not supported by clinical or experimental evidence—in contrast to the diabetic and tetanic (hypoparathyroid) cataracts.

An alternative explanation, based on the comparison of the clinical and the experimental dystrophic syndromes, will be attempted in the following section.

Distribution of Manifestations of Experimental and Clinical Dystrophies with Cataract.—The most important unifying feature in the group of dystrophic cataracts is the association of these cataracts with lesions in the cornea, skin and testes (table; columns 3, 4 and 5). In contrast, the dystrophies that involve chiefly the central nervous system and those that involve chiefly the hemopoietic system are not generally complicated by cataract.

The similarity of the distribution of manifestations in the syndromes of the dystrophic group suggests a metabolic relation both of the affected tissues and of the different syndromes. It may be suggested, for example, that the affected tissues resemble one another in some aspect of their metabolism and that this common metabolic process is interrupted at various related points in the different syndromes. For this reason, it is of interest that a complex of manifestations similar to that of the clinical dystrophic syndromes can be produced experimentally by diets deficient in certain chemically known vitamins or vitamin-like substances.

Role of Heredity and Environment in the Dystrophies with Cataract.—There is evidence that in all the clinical forms of dystrophic cataract an intrinsic or genotypical factor conditions the development of the lesion (table, last column).

At first sight, the attempt to group these idiopathic, endogenous conditions with those exogenous conditions produced experimentally may seem forced. Such a conception, however, is not new and is well illustrated, for example, in the analysis of the intrinsic and extrinsic factors in pernicious anemia and in the relation between pernicious anemia and tropical sprue.[79] Another example is the relation between exogenous and endogenous rickets (Eliot and Park [80]).

It is today a generally accepted biochemical concept that enzymatic (particularly oxidative) processes in the tissue require two main units: enzymes and coenzymes, the former being proteins, and thus likely to show genotypical (hereditary) characters,[81] while the latter are vitamins or vitamin-like compounds, which are not, or are not entirely, proteins. That the same group of tissues may be affected either by the absence in food of an exogenous (extrinsic) factor or by a genotypical defect of the endogenous (intrinsic) part of the same metabolic system does not appear surprising and is compatible with modern biochemical concepts.

At present the possibility can only be suggested that some metabolic link exists between the endogenous and the exogenous forms of dystrophic cataract. In the present paper the similarity in the clinical patterns of certain exogenous and certain endogenous cataract syndromes has been pointed out. It is to be hoped that this may serve, at least partially, to delimit the field in which such metabolic links may

79. Strauss, M. B., and Castle, W. B.: New England J. Med. **207**:55, 1932.

80. Eliot, M. W., and Park, E. A.: Rickets, in Brennemann, J.: Practice of Pediatrics, Hagerstown, Md., W. F. Prior Company, Inc., 1940, vol. 1.

81. Wright, S.: Physiol. Rev. **41**:487, 1941.

be sought. Still other exogenous dystrophic syndromes with cataract which may be discovered in the future may have to be considered similarly in relation to the endogenous syndromes.

In this concept, emphasis is placed on the hereditary basis of primary metabolic disturbances in the tissues of the affected organs in the endogenous dystrophic syndromes. It appears that such a concept is in better agreement with the clinical and experimental observations in cases of dystrophic cataract syndromes than a theory which locates the primary disturbance only in one or several special endocrine glands.

SUMMARY

On the basis of a previously suggested classification of experimental cataracts in the rat and of a bibliographic review of experimental and clinical "metabolic" cataract syndromes, three groups of cataracts are differentiated: the diabetic (hyperglycemic), the tetanic and the dystrophic. Morphologic and developmental features of the cataracts, as well as the allied symptoms, served as criteria for this differentiation.

The similarity in the clinical pattern between the clinical members of the dystrophic group and certain exogenous experimental dystrophic conditions is pointed out. The possible significance of this similarity for the metabolic interpretation of the genotypical dystrophic syndromes with cataract is discussed.

The assumption of primary metabolic disturbances in the affected tissues on a hereditary basis accounts better for the known observations than does the theory of a primary endocrine basis of the dystrophic syndromes.

THE FITTING OF CONTACT LENSES

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The fitting of contact lenses has been more or less a trial and error procedure. There is no doubt that the molded plastic lens is easier to adjust than the old type glass lens. The difficulty has been in knowing what adjustments are necessary in order to fit the individual eye. After observing thousands of contact lenses, we have worked out a scientific method of adapting these devices which has proved to be most satisfactory. Simple rules have been formulated which may be applied in every case. They eliminate guesswork and give complete assurance and confidence that the lenses will be fitted correctly and skilfully, according to plan.

PROCEDURE

In the fitting of contact lenses one must bear in mind the following factors: (1) convergence of the eyes; (2) pressure of the lids, and (3) gravity drop.

To compensate for these factors, a contact lens should be fitted so that the corneal portion will be turned slightly nasally and decentered upward.

A perfectly fitting lens is one that rests evenly on the sclera without impeding the blood vessels. No tight areas, or areas of blanching, should appear in any of the possible movements of the eye. On the other hand, there should be no loose areas beyond the corneal section.

The fitter should adhere to a definite routine in adapting the contact lens to the patient's eye. The following cardinal steps are to be observed in fitting the semi-finished lens: 1. The lens is checked for over-all size. 2. The lens is checked for rotation. 3. The fluorescein test is applied to detect (*a*) loose and (*b*) tight areas. 4. The corneal center of the lens is marked. 5. A working diagram is made.

STEP 1.—Sometimes a contact lens is so large that it cannot be inserted. The size is reduced from the upper portion of the nasal margin downward and along the lower border to the temporal edge. The upper scleral portion is not disturbed unless it is absolutely necessary.

Before the lens is inserted, a horizontal line is drawn from the outer to the inner canthus of the lens with a red china-marking pencil, Blaisdell 165 T. The lens is inserted with a clear solution. If it is impossible to see the nasal and lower edges of the lens while the eye is looking temporally and upward respectively, the lens is probably too large. The lower portion of the lens for the right eye is trimmed from 2 to 9 o'clock, and the lower part of the lens for the left eye, from 10 to 3 o'clock. The lens must clear the semilunar membrane and the caruncle.

Likewise, if the temporal edge of the lens interferes with the motion of the eye by striking against the outer fornix, the lens is too large. The exact area of interference is marked, and the edge is trimmed so that the lens moves in unison with the eye.

STEP 2.—Any rotation of the lens is easily observed, as the horizontal line on the lens will not remain stationary. The fitter's first consideration is to stop the

rotation by relieving excessively tight areas or by tightening loose edges. Before these areas are marked on the lens, it is made certain that the lens is in the proper position. If necessary, the lens is turned with the suction cup or the finger and is held in place. Then the exact areas which need adjustment are marked. Adjustments are repeated until the lens maintains a fixed and correct position in the eye.

STEP 3.—Two drops of a 1 per cent solution of fluorescein sodium is placed in the contact lens, and the lens is filled with solution and inserted. A cobalt blue or an ultraviolet ray filter is employed in observation of the lens. The fluorescein glows with a brilliant yellow-green reflex wherever the corneal section of the lens does not touch the cornea. Where the lens is in contact with the cornea a black area will be seen. All loose areas, which are indicated by the pools of fluorescein, and all tight areas, which are evident as blanched spaces, are marked on the lens. To observe tight areas it is necessary to use a white light.

The blanching is caused by the stoppage of blood flow in the vessels. It is vitally important that only areas needing adjustment be marked. The markings should not be extended beyond the limits of the exact area requiring correction.

STEP 4.—While the lens is in the eye, a mark is placed on the corneal portion directly in front of the center of the pupil. By marking the pupillary center of the eye one can determine its relation to the optical center of the lens.

STEP 5.—We suggest that the following procedure be adopted as a routine: A blue print–like record of the corrective markings is made on a diagram. This is an indispensable aid to the fitter and not only serves as a permanent record but can be used as a basis for analysis and for planning the necessary adjustments.

The actual visualization of the lens on paper will be of great aid to the fitter in determining the course of action. Further, a clearly defined picture of the patient's lens can be used as a valuable means of comparison by any fitter making future adjustments.

Before elimination of either tight or loose areas, the lens should be divided into four quadrants. This procedure will enable the fitter to plan his corrections and to apply the suitable method of procedure, as outlined in the rules and diagrams in the subsequent sections of the paper.

It will be noted that these rules call for relief of either the temporal or the nasal side first, as the case may be. This means that the entire temporal or the entire nasal half of the lens, as divided by the vertical line, must be adjusted. In other instances, it will be noted that correction is called for in either the upper or the lower portion. Again, this indicates that the entire portion of the lens above or below the horizontal line must be adjusted.

In the ideally fitting lens, the pool of fluorescein is sharply defined, and there is no infiltration into the scleral portion, as indicated by the absence of loose areas. There are no pressure, or tight, areas on the sclera, as evidenced by the absence of blanched patches. However, it is not always essential to achieve this ideal result.

CORRECTION OF TIGHT, OR PRESSURE, AREAS

For relief of areas of pressure the inner scleral surface of the involved area is ground with a grit-impregnated rubber point. The rubber point is fitted on a dental mandril, inserted in the hand piece of a dental lathe. A light rotary motion is used. Pressure is not exerted, but the tool is allowed to do the grinding. All ground areas are repolished with a felt disk and a polishing compound before the lens is reinserted. All tight areas are to be eliminated before the loose areas are adjusted.

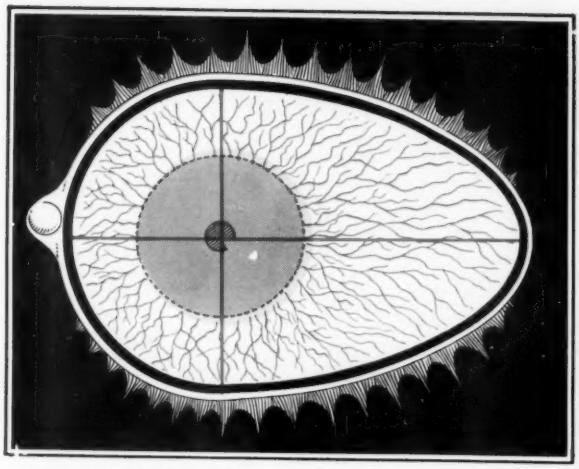

Fig. 1.—Division of the lens into four quadrants.

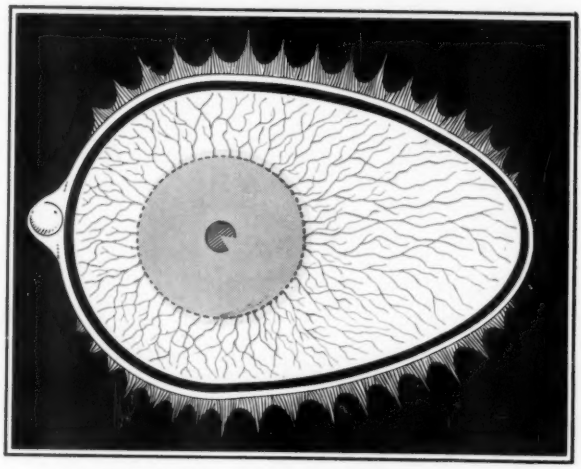

Fig. 2.—The ideally fitting lens.

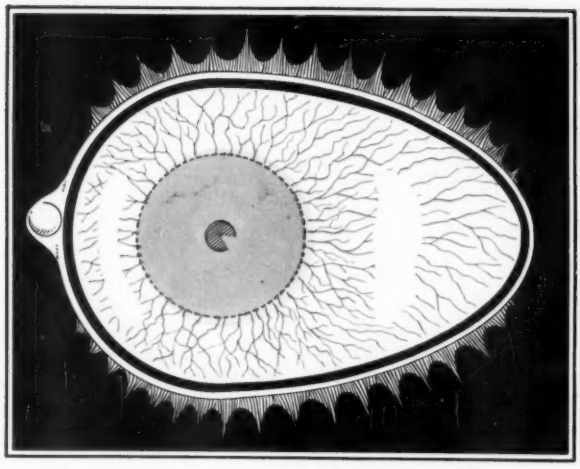

Fig. 3.—Tight areas in the nasal and temporal portions of the lens.

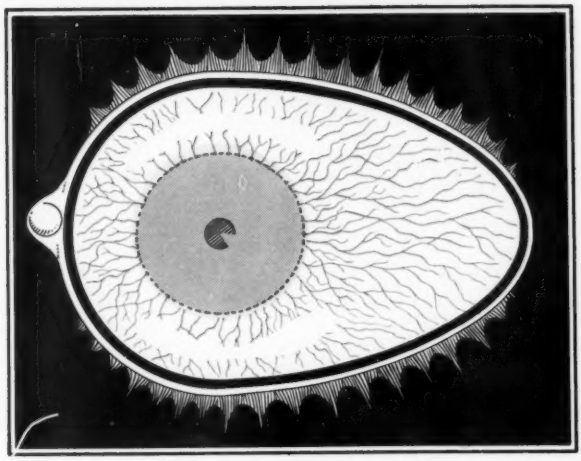

Fig. 4.—Tight areas in the lens above and below the horizontal line.

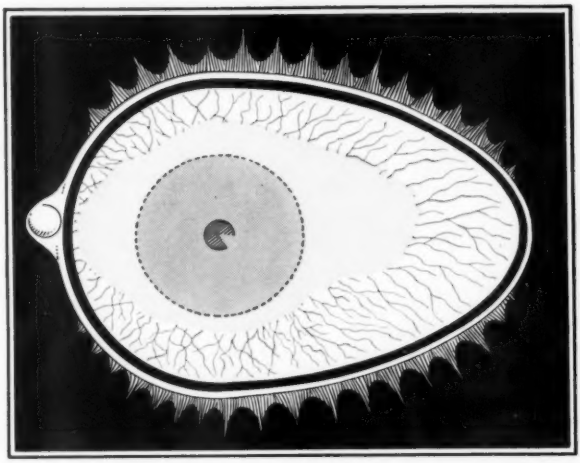

Fig. 5.—Fluorescein pools beneath the nasal and temporal portions of the lens.

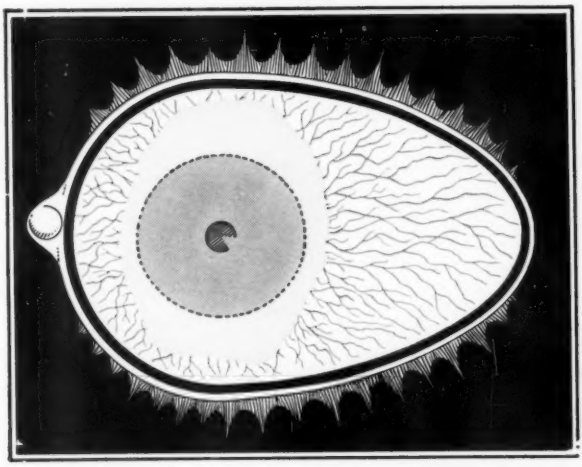

Fig. 6.—Fluorescein pools above and below the horizontal line of the lens.

When he is making adjustments or corrections, the fitter should be guided by the following rules:

RULE 1.—If the lens is tight nasally and temporally, pressure is relieved on the temporal side first, except when the optical center is to the nasal side of the pupil, in which case pressure on the nasal side is relieved first. Pressure on the temporal side is relieved first in order to allow the lens to slide nasally and to prevent it from moving out of its proper position. Often when the temporal side is loosened, the area of pressure on the nasal side disappears. If the optical center of the lens lies to the nasal side of the pupillary center, then pressure on the nasal side is relieved first. The tight area on the temporal side will cause the lens to shift temporally. The optical center of the lens will finally coincide with the center of the pupil.

RULE 2.—If the lens is tight above and below the horizontal line, pressure in the lower portion is relieved first, except when the optical center is higher than the pupillary center, in which case the upper portion is corrected first. Pressure in the lower portion is relieved first in order to allow the lens to slide upward. The pressure above the line will be lessened. If it does not disappear altogether, the corresponding area of the lens should be ground, but only after the lower area of pressure has been adjusted. Pressure in the upper portion is relieved first only when the optical center is above the pupillary center. If the upper portion is ground, the lower area of pressure will force the lens downward, and the optical center will coincide with the pupillary center.

Any isolated tight areas should be adjusted by the usual methods. When the tight areas are corrected, many of the loose areas will probably disappear. However, it is necessary to make a second fluorescein test for detection of any remaining loose areas.

CORRECTION OF LOOSE AREAS

Corneal contacts are caused by loose areas. If the lens does not rest evenly on the sclera, the loose areas allow the eye to move behind the lens, with the result that the lens strikes the cornea. Loose areas are detected by the infiltration of the fluorescein solution into the scleral portion, with the formation of pools. These pools must be eliminated when a corneal contact is present. If no corneal contact is observed, the loose areas need not be adjusted.

Elimination of the pools is effected by grinding of the portions of the inner surface of the sclera that do not show the presence of fluorescein—in other words, the regions surrounding the loose areas. This allows the lens to settle back, with the result that the pools are reduced in size and finally disappear.

RULE 3.—If there are corneal contacts both nasally and temporally, the nasal pool is eliminated first, except when the optical center is to the nasal side of the pupil, in which case the temporal pool is eliminated first. By elimination of the loose areas at the nasal side first, the lens is allowed to settle at the nasal side, so that it is kept in its proper position, i. e., turned a little nasally, to allow for convergence. The loose area at the temporal side generally decreases in size, and, if necessary, the inner surface of the lens around this area may be ground.

RULE 4.—If there are corneal contacts both above and below the horizontal line, the upper pool is eliminated first, except when the optical center is higher than the pupillary center, in which case the lower pool is eliminated first. If the upper pool is eliminated first, the lens will settle into position, so that the slight drop due to gravity and the pressure of the lids are offset by an increase in the bearing surface of the upper part of the sclera.

Other Procedures.—Rarely is there a corneal contact below the line. However, if one is present or the optical center is above the pupil, then the portion of the lens surrounding the lower pool is ground away. The lens will fit closer to the lower part of the sclera, and the optical center will move downward.

In cases of corneal contacts not covered by rules 3 and 4 the following procedures are in order:

1. When a single contact occurs, the region around the adjacent loose area is ground away.

2. At times a contact lens will seem to have a clearly defined fluorescein pool, as illustrated in the ideally fitting lens, and yet a corneal contact may appear. In such a case if the lens is moved gently in the direction in which the corneal contact appears, a slight infiltration of fluorescein into the scleral section will be noted. This indicates a loose area. This area should be marked and the portion surrounding it ground off until the lens settles in position, with elimination of the corneal contact.

3. When the periphery of the lens stands away from the sclera, the edges should be tightened. This is accomplished as follows: The lens is placed over the cast (dental stone positive); the loose area is marked on the cast, and then some of the dental stone is removed with a knife and the marked area smoothed with sandpaper. The lens is replaced on the cast. A dental wax spatula is heated over an alcohol lamp. The tool is placed on the loose area of the lens, and a rocking motion is used. The tool is not removed until it has completely cooled. This procedure is repeated until the loose area has been eliminated. The tool is not allowed to become too hot, as it will soften the material.

49 East Fifty-First Street.

NEURODERMATITIS WITH CATARACT

REPORT OF TWO CASES

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Prior to 1921 there were no American reports on the syndrome of cataract associated with neurodermatitis. In the European literature, however, Rothmund,[1] in 1868, described the occurrence of cataract in connection with changes in the skin. Andogsky [2] used the term "cataracta dermatogenes" in describing a case of bilateral cataract with an associated cutaneous eruption. Similar cases have been reported by Löwenstein,[3] Gault,[4] Winkler [5] and others.

The first report in the English language was that of Davis,[6] who, in 1921, described the case of a 15 year old girl with neurodermatitis accompanied by cataracts, which matured in one year. Daniel,[7] from the Mayo Clinic, in 1935, reported 3 cases and mentioned the importance of a family history of allergy.

At first it was generally considered that the "neuro" part of the name alluded, perhaps, to some disturbance along the course of the nerves; actually, it refers to the unstable sympathetic system and emotional background of the patient. The papular rash is distributed over the face, neck, chest and flexor surfaces. The skin becomes ichthyotic and thickened, as though covered with a layer of shellac. Intense itching is the chief symptom. The endocrine system frequently seems to play a definite etiologic role. Epinephrine, thyroid, parathyroid and pituitary extracts have been reported of value in treatment in some cases. Brunsting [8] stated:

> Those who wish to preserve the concept of a primary nervous instability as an important part of the background in cases of these disorders still cling to the term "neurodermatitis."

He further stated:

> One cannot deny that the elements of chronic fatigue and nervous imbalance must be considered in the proper control of this disorder, but it does not seem likely in my opinion that the nervous background is primarily responsible for the syndrome.

He apparently believed in the existence of a certain allergic factor, and this opinion was strengthened by the frequent family history of allergy and by the fact that of his 101 cases, cataract was present in 10 and the average eosinophil count was slightly over 9 per cent.

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1. Rothmund, A.: Ueber Cataracte in Verbindung mit einer eigentümlichen Hautdegeneration, Arch. f. Ophth. (pt. 1) **14**:159-182, 1868.

2. Andogsky, N.: Cataracta dermatogenes; ein Beitrag zur Aetiologie der Linsentrübung, Klin. Monatsbl. f. Augenh. **17**:824-831, 1914.

3. Löwenstein, A.: Katarakt bei Neurodermatitis, Klin. Monatsbl. f. Augenh. **72**:653-657, 1924.

4. Gault, N.: Cataracte névrodermitique, Bull. Soc. d'opht. de Paris, 1933, pp. 280-284.

5. Winkler, A.: Beitrag zur Genese der Cataracta dermatogenes, Arch. f. Ophth. **139**: 526-531, 1938.

6. Davis, W. T.: The Relation of the Eye and Certain Skin Diseases, South. M. J. **14**:237-241, 1921.

7. Daniel, R. K.: Allergy and Cataracts, J. A. M. A. **105**:481-483 (Aug. 17) 1935.

8. Brunsting, L. A.: Atopic Dermatitis (Disseminated Neurodermatitis) of Young Adults: Factors in One Hundred and One Cases and Report of Ten Cases with Associated Cataracts, Arch. Dermat. & Syph. **34**:935-957 (Dec.) 1936.

The following 2 cases illustrates the syndrome of neurodermatitis associated with cataract.

REPORT OF CASES

CASE 1.—A white youth aged 18 years was first examined on March 22, 1937. He had complained of poor vision in both eyes for one year.

Family History.—His mother had suffered from Ménière's disease for the past five years; his father was living and in good health. He had 1 sister, aged 10 years, who was healthy.

Personal History.—Birth was normal. Asthmatic attacks began at the age of 5 months and continued to recur for ten years. Eczema appeared at the age of 5 years. He had the usual diseases of childhood, as well as two attacks of otitis media. He stated that he was extremely allergic. During the entire time he was under my care he proved to have a decidedly emotional temperament.

Examination showed that vision in each eye was 20/65, unimproved. Five weeks later this had decreased to ability to see hand movements with the right eye and to 20/200 in the left eye. The eyelids were brawny; the conjunctivas were normal. Each eye showed anterior and posterior cortical opacities in the lens, a clear nuclear area remaining. The reflex from the fundus of each eye was pink, with imperfect details. Tension was normal in each eye.

In May 1937, an extraction of the cataract was performed on the right eye through a subconjunctival stereocorneal incision made with a keratome. The lens proved to be soft throughout. No sign of a nucleus was observed. Recovery was uneventful. Corrected vision was 20/13.

By July 1937, vision in the left eye had decreased to counting of fingers at 2 feet (60 cm.). On Oct. 25, 1937 the cataract was extracted by the method already described. The result was corrected vision of 20/13.

When the patient was last examined, on Oct. 14, 1942, corrected vision in each eye was still 20/13. The cutaneous condition had improved, there being less itching and redness.

CASE 2.—A white woman aged 23 was first examined on July 23, 1937. She had been told six or seven years ago that cataracts were developing. She stated that for four or five years she had received roentgen therapy. These treatments were discontinued two years prior to my seeing her. During the latter period glandular extracts and concentrated vitamin preparations were administered.

Family History.—There was a history of nervous instability in both parents.

Personal History.—When the patient was 11 years old, eczema first appeared under the arms, resembling multiple boils. In the course of one year a similar lesion broke out on the neck and face. Asthmatic attacks began at the age of 13 years and still continued. The usual diseases of childhood occurred. Many tests for allergy showed a positive reaction. Her menstrual cycle was always normal except for one period of suppression during the roentgen treatments.

Ocular Examination.—Right Eye: The lids were thickened, brawny and ichthyotic. The conjunctiva was slightly congested. There was a keratoconus with a dense opacity in the lower quadrant, due to breaks in Descemet's membrane. The anterior chamber was deep. From its milky appearance the cataract appeared to be hypermature. Vision was limited to ability to see hand movements.

Left Eye: The lids and conjunctiva showed a picture identical with that in the right eye. There was also a keratoconus with a dense opacity, due to breaks in Descemet's membrane. The cataract was immature. No details of the fundus could be observed. Vision was 20/100, unimproved.

Tension in both eyes, taken with the fingers because of the keratoconus, was high normal. This was reduced to normal by use of pilocarpine.

Operations.—On April 3, 1939 a linear extraction was performed on the right eye, the same method being employed as that described in case 1. The cataract was found to be very soft throughout, so that it was readily removed by irrigation. Recovery was uneventful. Corrected vision was 20/50; that it was not better was due to the keratoconus.

On April 8, 1942 a similar linear extraction was performed on the left eye. Recovery was somewhat delayed, probably owing to an exacerbation of the eczema elsewhere in the body. On the last examination, Oct. 23, 1942, corrected vision was 20/65, a little less than that in the fellow eye, because the corneal scars were denser.

SUMMARY

1. The patients whose cases are reported were both emotionally unstable and reacted positively to many different tests for allergy.

2. The skin of the eyelids of each patient was brawny, red and itchy, with no involvement of the cilia, as has been reported by some observers.

3. The conjunctiva was not involved except in 1 case, in which it was slightly inflamed.

4. Both the patients were young persons whose cataracts developed during the active stage of eczema and neurodermatitis.

5. Both patients belonged to the white race.

6. In each patient, after the first definite evidence of opacities in the lenses, the maturing process was rapid.

7. There was no undue postoperative reaction, except for the trivial effect in one eye, as previously explained.

CONCLUSIONS

The absence of undue postoperative reaction after the removal of cataracts from both eyes of 2 patients in the active stage of neurodermatitis seems to indicate that such a condition lends itself well to surgical intervention and presents no more hazards than does cataract complicated by other diseases.

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RETINAL DETACHMENT AND TRAUMA

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As Parsons succinctly put it, "The exact mechanism of detachment is not completely understood; indeed detachment of the retina is still one of the difficult problems of pathology." In other words, as it is not known how a detachment is produced, its relation to trauma must remain vague. Yet one cannot but be struck by how generally the thesis of a traumatic origin is accepted. For some time I have noted, in examining patients with retinal detachment, how rare a traumatic origin really is, and this has led me to review 400 successive cases in which I have had the opportunity to make examination.

These 400 cases may be classified as follows: [1]

Associated Condition		No. of Cases	Percentage
Myopia		202	50
Trauma		52	12
Direct	32		
Indirect	20		
Hemorrhage (vitreous and retinal)		21	
Aphakia		37	9
Extracapsular extraction	23		
Intracapsular extraction	7		
Discission	7		

Cases of bilateral detachment numbered 58, or 14 per cent.

The cases of myopia were represented in the etiologic groups as follows:

Etiologic Condition	No. of Cases	Percentage
Trauma		
Direct	10 in 32	33
Indirect	15 in 20	75
Aphakia		
Extracapsular extraction	11 in 23	45
Intracapsular extraction	1 in 7	14
Bilateral	32 in 58	55

The degree of myopia was over 10 D. in 25 per cent, from 5 to 10 D. in 42 per cent and up to 5 D. in 33 per cent of cases. The danger of detachment gradually increased with the degree of myopia up to a certain point and was greatest between 8 and 16 D. of myopia. Detachment occurred with the greatest frequency in persons between 45 and 60 years of age, when nutritional disturbances occur in the ocular tissues. The incidence of trauma decreased as the degree of myopia increased. Direct injury was more frequent in cases of hypermetropia than was indirect injury. Spontaneous detachment was the rule in cases of myopia.

It seems reasonable to divide cases of traumatic detachment into those in which the detachment occurs in previously healthy eyes and those in which it occurs in previously defective eyes and to classify the traumas as direct and indirect. The

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1. The figures in the tabulation do not add up to 400, as the number of cases of hypermetropia was omitted, and in a considerable number of instances the refraction was not indicated. The real number of cases of myopia was unquestionably much greater than 202.

types of trauma in this series of cases were the same as those usually indicated in the literature.

Direct trauma generally resulted from direct blows to the eyeball with a ball, fist, stone or other hard substance. It occurred principally in cases of young persons. Myopic eyes did not preponderate. The types of lesions were the oral tear and round or giant holes, but the horseshoe-shaped tear was not observed. Although detachment of the retina can best be explained by contrecoup, choroidal and scleral ruptures were apparently caused by different, and probably more severe, injuries. Perforating injuries were excluded, as the detachment then results from lesions of the vitreous following hemorrhage or inflammation.

Indirect trauma was caused by severe blows to the head or body; succussion of the head or body; excessive bodily exertion, such as that in lifting, and excessive coughing or sneezing. The injury might be mild, such as that produced by rubbing the eyes with the fingers, long motor or railway journeys, fluid splashed in the eye, extraction of a tooth or removal of a foreign body from the cornea. Indirect injuries are in themselves vague, and the length of time between the alleged trauma and the onset of the detachment is indefinite. In my series of cases of indirect trauma the patient's age was generally above 40; the 2 youngest patients were each 29 years old. The refraction showed myopia in 15 cases and hypermetropia in 4 cases. The type of retinal tear was not characteristic, but there were no oral tears.

There can be no question about the traumatic origin of a detachment in young persons with normal eyes who have suffered severe injury to the eyeball and that of a detachment which follows a penetrating injury of the eyeball. The role of indirect injuries is more difficult to explain, especially the mild ones. To explain the relation between retinal detachment and indirect trauma a predisposition on the part of the eye is assumed, and it is believed that in the presence of certain changes in the eye (predisposition), a mild injury or a sudden circulatory disturbance on muscular exertion may be the determining cause of a detachment which is ready to develop, just as the detachment generally occurs without trauma.

The predisposition consists in certain changes in the eye which are present in myopic eyes and make such eyes susceptible to retinal detachment. Among these, cystoid degeneration of the retina, first described by Leber and confirmed by Gonin, occurs in the external plexiform layer, with changes in the pigment epithelium, implying that the choroid is also affected. Clinical signs of inflammation are absent, and a local circulatory disturbance must be considered a factor. Vogt showed that similar changes occurred in the aging eye from nutritional disturbances, and Hanssen demonstrated tears and other changes in the retina in myopic eyes without detachment of the retina.

The vitreous, also, takes part in this degeneration and undergoes fluidification, shrinkage and detachment. Gonin, in his microscopic study of eyes with retinal detachment, found that the vitreous was always shrunken from one third to one half of its former size, and this observation has been confirmed by other investigators (Lister, Lindner and von Sallmann). The presence of a detachment of the vitreous has been recognized clinically by several investigators (Benziger, Pillat, Lindner, von Sallmann and Vogt).

Adhesion of the vitreous framework to the retina occurs in healthy eyes at the base of the vitreous and has been observed on histologic examination in places in which the vitreous is adherent to the retina, where the greatest changes in the retina exist and where the hole in the retina is situated. The presence of this adhesion to the margin of the hole has been observed on clinical examination with the slit lamp by Böck.

Finally, as the vitreous body was known to move with movements of the eyeball (Best), it seemed logical to a number of investigators that this movement can easily cause traction on the retina and that if the retina is weakened a tear results and conditions necessary for the development of the detachment are present (Leber, Gonin, Lindner and others).

If the same histologic changes explain the onset of spontaneous and of traumatic detachment, it is impossible to draw a sharp line between the two types. If movements of the vitreous are accepted as a factor in the causation of spontaneous detachment, they can, all the more, explain the mysterious detachment after an indirect trauma. Anything, therefore, which will cause movements of the vitreous can produce a detachment in a predisposed eye. But Lindner showed in his experiments that it is not just a shaking of the eyeball that produces a detachment but that the force must be of a rotating or whirling nature. The question arises: Under what conditions does the eyeball undergo ocular rotation? It has been suggested that with indirect trauma the patient instinctively makes a rapid protective movement of the eyes, whereby the vitreous drags on the upper half of the retina and causes a rupture of the retina.

Krückmann [2] reported the cases of 3 young persons with emmetropia who were carrying heavy weights; the weight slipped; the body straightened, and the eyes were forced upward and strongly abducted. On the following day a detachment was present, in the lower temporal quadrant, which the author explained on the basis of retinal swelling, following sudden ischemia, similar to cerebral swelling. The location is explained by action of the inferior oblique muscle, which suddenly contracted when the eyes were raised. This produced aspiration of the sclera with vacuum action on the retina. Krückmann also considered the role of muscular action in the development of detachment in cases of indirect trauma. The muscles that came under consideration were the two oblique muscles, as these are the only muscles whose insertion on the sclera covers an area of the sclera and choroid beneath which retinal tissue is situated directly. In the aforementioned cases the inferior oblique muscle was regarded as the active factor. This condition, according to Krückmann, differs from the tears in the retina which occur in the insertional area of the superior oblique muscle in the myopic eye and are explained by uneven traction of that muscle because of its peculiar anatomic structure and insertion.

Bartels [3] drew attention to the role of the superior oblique muscle in explaining the prevalence of retinal holes in the superior temporal quadrants.

MacDonald [4] suggested that a hydrodynamic phenomenon may cause detachment of the retina in persons who do heavy lifting. The transmitted intracranial pressure obstructs the central retinal vein, and the excess fluid from the choroidal exudate and the central retinal artery collects in the retina and the intraretinal space, with resulting detachment.

Walker [5] raised the interesting question why detachment occurred in only one eye when the injury was general and gave a reason for the frequency of retinal tears in the upper temporal quadrant. This frequency the author explained by the statement that the retina in the upper temporal quadrant is under additional mechanical disadvantage, pressure and vacuum action, as the superior oblique

2. Krückmann, E.: Netzhautablösung und indirektes Trauma, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **51**:304, 1936.

3. Bartels, M.: Ueber die Enstehung von Netzhautablösungen, Klin. Monatsbl. f. Augenh. **91**:437, 1933.

4. MacDonald, A. E.: Etiology of Idiopathic Retinal Detachment, Am. J. Ophth. **21**:658, 1938.

5. Walker, in discussion on MacDonald,[4] p. 661.

muscle is exposed to peculiar punishment, which is greatest in the eye acted on most strongly by its superior oblique muscle in the conjugate positions.

Friedman,[6] in an article on traction on the optic nerve as a force in production of retinal detachment, spoke of (1) the movements of the vitreous in rapid movements of the eye as related to the phenomenon of inertia and (2) pressure on the globe by the extraocular muscles, which is transmitted to the retina. The latter factor was suggested by the frequency of tears in the region underlying the superior oblique muscle, which is being constantly used in all close work. The author concluded that both these factors act on the retina during ocular rotation and that the use of stenopaic goggles is sound.

Perhaps when more is known about detachment of the retina, the problem of its relation to indirect trauma will be explained. There are still many problems concerned with this subject. With the appreciation of the significance of the retinal hole other important features may be overlooked. Some investigators, especially Kümmell, have claimed that it is not the hole alone which causes detachment but that changes in the hydrostatic equilibrium between the preretinal and the retroretinal spaces, from reduction in vitreous pressure, constitute an important contributing factor and should not be disregarded. Attention has repeatedly been drawn to the fact that in many cases contusion does not lead to detachment even in myopic eyes and that detachment is not observed after strenuous gymnastic exercises or in association with fractures of the skull or war injuries, and that while severe bodily exertion is common, resulting detachment is unusual. Though the cause of the detachment after indirect trauma is difficult to define, the question of the relation cannot be dismissed as irrelevant when such careful observers and scientific investigators as Gonin, Vogt and Jeandelize are firm believers in the role of indirect trauma. At the same time, all persons are inclined to find a cause for an accident, especially if compensation is a factor. A guiding rule in the estimation of the cause would be that the exertion must be greater than usual and that the symptoms of detachment should promptly follow. Davidson,[7] who had a large experience with compensation claims, stated that the association of trauma and detachment could be accepted only when the visual disturbance was complained of within two days after the injury and the detachment developed before the end of two weeks.

In a review of this series of cases of detachment in their relation to trauma, two other points become apparent and are, I hope, worthy of mention. The first is bilaterality of retinal detachment. This was present in 58 cases, or about 14 per cent, in 32 of which, or 55 per cent, myopia was represented. The feature of bilaterality is significant and speaks against a traumatic origin. The cases of bilateral detachment can be divided into cases involving juveniles and cases involving myopic persons and the aged. While the detachments were bilaterally symmetric in the young, in the other patients there was no symmetry. The cause was general, and the important factor was the local condition in the eye. A careful examination of the fellow eye in all cases of detachment is always important (Sourdille).

The second point of significance is the association of detachment with aphakia. There is still a great deal to be learned about this relation, and I shall refer only briefly to it here. Retinal detachment in aphakic eyes is related to trauma only in its widest sense; as with perforating injuries of the eyeball, there are set in motion changes in the intraocular structure, especially in the vitreous, which after some

6. Friedman, B.: Mechanics of Optic Nerve Traction on the Retina During Ocular Rotation, Arch. Ophth. **25**:564 (April) 1941.

7. Davidson, M.: Netzhautabhebungen infolge indirekten Traumas, vom Standpunkt des Arbeitsunfallgesetzes, Tr. internat. Ophth. Cong. **4**:201-203, 1933.

time lead to detachment. The aforementioned predisposition and the loss of vitreous at operation are undoubted factors, though the quantity of the vitreous lost is not the essential feature.

Detachment of the retina was observed in 37 cases of aphakia, of which extracapsular extraction had been performed in 23, intracapsular extraction in 7 and discission in 7. No deductions can be drawn from these figures, especially with respect to the relative frequency of retinal detachment after extracapsular and intracapsular extractions. Detachment of the retina after operation for congenital or lamellar cataract (discission, or needling) occurred in 6 cases, and in 1 case the operation was performed for high myopia. Shapland [8] drew attention to the frequency of detachment after discission of soft cataract in a report on 50 cases of detachment associated with aphakia, in 22 of which needling for soft cataract was performed. The detachment occurred after an average interval of twenty-four and six-tenths years. Of 40 eyes with congenital cataract in Shapland's series, detachment occurred in 33. The bad prognosis he ascribed to progressive myopia, and no benefit resulted from operation for the detachment. Moore [9] gave it as his opinion that removal of a lens, whether by extraction or by discission, predisposed to the development of detachment of the retina, and this in no negligible degree. Detachment occurred more frequently after the needling operation for soft cataract, and this, he suspected, was because of the prospect for so much longer life after this operation than after the usual extraction for hard cataract. Moore was so impressed by the poor prognosis of the needling operation for young patients that he doubted the wisdom of removal of both lenses for lamellar cataract unless the opacity was so dense that the eye was of little value if the operation was not done.

10 East Fifty-Fourth Street.

8. Shapland, C. D.: Retinal Detachment in Aphakia, Tr. Ophth. Soc. U. Kingdom **54**: 176, 1934.

9. Moore, F.: Diathermy in Ophthalmology, Tr. Ophth. Soc. U. Kingdom **53**:487, 1933.

Clinical Notes

PLASTIC SHELL FOR USE IN THE SIMPLE EVISCERATION OF THE GLOBE

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After many years of experience with Harold Gifford's method of simple evisceration of the globe, the only disadvantage which I have noted, when proper indications are observed, is the occurrence of conjunctival chemosis. It will be remembered that the operation consists in an incision across the cornea and into the sclera at each side, removal of the ocular contents by an evisceration spoon and careful wiping of the inside of the scleral shell with sponges to remove all uveal tissue. The entire cornea is retained. No sutures need be employed, or, at most, one suture between the lips of the corneal wound to keep them from overlapping during the healing process may be used. No implant is employed. The normal attachments of the conjunctiva are not disturbed, and, as a result of the trauma of evisceration, fluid tends to collect within and beneath the conjunctiva. This

Plastic shell for use in the simple evisceration of the globe.

chemosis may be severe, to the extent of protrusion between the lids, a phenomenon alarming to the patient.

Various means have been tried to combat this complication, the most effective being the constant use of finely cracked ice in a rubber glove and a pressure dressing. To be effective the pressure dressing must be applied either constantly, which excludes the use of ice, or every night, while the ice is not in use. In spite of these measures, or because of the difficulties in carrying them out rigorously in practice, difficulty with chemosis continued to occur not infrequently.

Hence an attempt was made to exert even pressure on the conjunctiva by some form of shell. Several experiments resulted in the shell shown in the figure.[1] This device resembles in shape a shell prosthesis; it measures 24 by 20 mm. in frontal dimensions and has a curve 7 mm. in depth. The shell is made with either white or transparent plastic material, the latter having the advantage that the stump may be observed without removing the shell. The same shell will fit either eye, and one of the dimensions given has been found to fit nearly all patients without difficulty. This includes children as young as 8 years. With infants it would be necessary to employ a smaller shell. The shell is inexpensive and may be sterilized in alcohol or in Bard-Parker solution [2] so that it may be used by more than one patient.

The shell is inserted on the operating table at the end of evisceration, and ice is applied in the usual manner through a single layer of gauze during the daytime

From the Department of Ophthalmology, Northwestern University Medical School.

1. This shell is made by Mager and Gougelmann, 30 North Michigan Avenue, Chicago.

2. The composition of the Bard-Parker solution is as follows: ethyl alcohol, 67.8 per cent; methyl alcohol, 9.3 per cent; formaldehyde, 8 per cent, and water, 14.9 per cent.

for one week after operation. With the shell in place it is not necessary to apply pressure at night, since the pressure of the lids on the shell prevents the occurrence of chemosis. The shell is easily removed for dressings by the suction cup used for insertion of contact glasses, but in the case of children it has proved practical to leave it in place for two weeks, at which time the first prosthesis may be fitted. Use of the shell is continued usually for this length of time, as if it is removed sooner chemosis may develop. It is usually best to postpone the fitting of a permanent prosthesis for at least two months, since slight changes in the shape of the stump occur during this time.

In addition to preventing chemosis, the shell holds the sclera and the cornea in correct position, so that the tissues are allowed to shrink uniformly. The impression has been gained that the stump is more regular and a little larger when the shell has been employed than when pressure has been applied without a shell.

In addition to its use after evisceration, employment of the shell is of advantage after enucleation with an implant in Tenon's capsule, an operation which may be followed by considerable chemosis. The additional use of a pressure dressing for two weeks is considered of great importance, however, after any implantation, to prevent displacement of the implant.

720 North Michigan Boulevard.

BILATERAL METASTATIC CARCINOMA OF CHOROID

Report of a Case

GEORGE MARTIN MCBEAN, M.D., CHICAGO

Bedell,[1] in a report of a case of bilateral metastatic carcinoma of the choroid, stated that only 250 cases of this condition have been recorded. It is late to record such a case which I encountered in 1916, but because of its rarity it should be listed.

Miss G. D., aged 52, white, a schoolteacher, had had both breasts removed for carcinoma, with recurrence of the neoplasm about the right clavicle, for which she was undergoing roentgen therapy.

The right eye showed an area of episcleritis on the temporal side and the fundus a pale gray zone, about the size of the disk and slightly elevated, under the insertion of the external rectus muscle. There was a scintillating scotoma, but the field otherwise was not contracted. Two weeks later severe pain developed about the right eye and the right side of the head; a subconjunctival hemorrhage also appeared while she was in the hospital under the surgeon's care. He had ordered instillation of atropine. When I saw her the next day, the tension was elevated, and the media were too hazy to permit visualization of the fundus. The tension (Schiøtz) was plus 60. Miotic drugs were instilled, and a paracentesis was done. Dr. Cassius Wescott examined her with me in consultation, and later we did an iridectomy. Although tension was reduced, severe pain recurred; the eye became blind, and enucleation was done. Dr. E. V. L. Brown and Dr. Francis Lane, who studied the eye, observed the tumor under the insertion of the external rectus muscle.

Metastases occurred later in other regions of the body, including the femur of one leg, which underwent spontaneous fracture. Shortly before the patient died, I examined the left eye, which was becoming blind, and found a wide retinal detachment covering a solid growth. Enucleation was not permitted either before or after death, but I believe this eye was also the seat of a metastatic carcinoma.

25 East Washington Street.

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1. Bedell, A. J.: Bilateral Metastatic Carcinoma of the Choroid, Arch. Ophth. **30**:25 (July) 1943.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

LOCAL ANESTHESIA IN OPHTHALMOLOGY

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The aim of this article is to assemble and make easily available the more generally accepted methods of local anesthesia in ophthalmic practice. A few modifications that have been tried and found satisfactory are included.

HISTORICAL REVIEW

Anesthesia has unquestionably been one of the great factors in the evolution of modern surgery and is, in a large measure, responsible for the tremendous advance that has been made in surgery since 1846, when it was introduced. It is difficult to picture surgery without the aid of anesthesia, but this was the situation less than one hundred years ago. In spite of the fact that the desire to prevent pain during surgical operations dates back to antiquity, this aim was not realized until comparatively recently.

The early Egyptian, Chinese, Greek and Roman physicians attempted to allay the pain incident to surgical operations by the use of alcoholic beverages, drinks with narcotic properties and inhalations of narcotic drugs. In medieval times the *spongia somnifera,* or "sleep sponges," were very popular and were rather generally used in an attempt to produce surgical anesthesia. In the "Antidotarium" of Nicolaus Salernitanus an interesting account of these sponges is given:

> Take of opium of Thebes one ounce, juice of hemlock, poppy capsules, juice of mandragora, juice of wood-ivy, of each, one ounce. Put all these into a vessel together with a new sponge which is just as it came from the sea and has not been in contact with fresh water. Place the vessel in the sun during the dog-days until the sponge is needed, moisten it with a little hot water and apply it to the patient's nostrils, and he will quickly fall asleep. When you wish to rouse him, apply the juice of fennel-root, and he will soon wake.

This prescription expresses all the confidence of modern advertisements. The use of these "sleep sponges" was gradually discontinued because in doses sufficiently large to produce narcosis the drugs were distinctly dangerous and in smaller quantities the results were disappointing. Also, as suggested in the "Bibliotheca Osleriana," the sponges were abandoned partly because of the introduction of the tourniquet for amputations and operations on the extremities.

The ophthalmologic portion of the "Tetrabiblion" of Aëtius of Amida (502-574 A. D.), which covers rather fully the ophthalmology of the ancients, makes no mention of any attempt at anesthesia in surgical operations. In chapter LXVIII, in discussing the operation for trichiasis, he commented: "As, however, a few patients, because of cowardice, can never bring themselves to take an operation . . ." This, with similar references, leads one to believe that, in lieu of anesthesia, the ancients relied principally on stalwart assistants to secure a quiet, if not a relaxed, patient. That the patients were not always quiet is evident from another statement, regarding the operation for pterygium, in which Aëtius remarked that care must be observed in order to avoid cutting the lids.

This review is a revision of a candidate's thesis for membership in the American Ophthalmological Society (Local Anesthesia in Ophthalmology, Tr. Am. Ophth. Soc. **32**:399, 1934).

In early times suggestion or hypnotism probably accounted in a measure for the successful relief of pain by surgeons. This was often combined with some particular procedure for supposed anesthesia, comparable in merit to that of placing the fat of the crocodile or its powdered skin on the patient's skin, a practice of the early Egyptions. Also, according to Plinius, the "stone of Memphis" produced local anesthesia if it was mixed with vinegar and rubbed on the skin. The various methods of securing local anesthesia advocated from time to time were tried by enthusiastic followers and later discarded by them, since they probably lacked the same hypnotic ability as the advocates of the methods.

Pressure on the nerve trunks, particularly of the extremities, to produce anesthesia was used as late as 1870, but it was never a popular procedure, since sufficient pressure to cause insensibility of the part supplied by the nerve was frequently painful and required considerable time and satisfactory anesthesia was not often obtained. In addition, there was grave danger of pressure necrosis or permanent motor and sensory paralysis. This method was too uncertain, and the danger was too great. Cold as a means of producing local anesthesia was first introduced in the middle of the sixteenth century by Thomas Bartholinus. Various agents, such as ice, ice with salt, ether spray and other rapidly evaporating fluids, have been used. In minor surgery carbon dioxide, ethyl chloride and ethyl bromide are still occasionally employed to freeze the tissues and to produce local superficial anesthesia. Repeated attempts to induce local anesthesia by the application or injection of sleep-producing drugs were made on the erroneous theory that a similar effect would be produced locally.

A discovery of considerable importance, which made possible the present methods of local anesthesia, was the hollow needle for hypodermic injections, introduced by Alexander Wood, of Edinburgh, Scotland, in 1853. This important mechanical aid made it possible to introduce drugs into the tissues and to bring them in direct contact with the nerves supplying the part, so that when drugs that would induce local anesthesia were discovered, a means of using them to the best advantage was available.

It is hard for surgeons in this generation to conceive of the possibility, with their highly sensitive, nervous patients, of performing even the simple removal of a foreign body from the cornea without the assistance of a local anesthetic, and extraction of a cataract without the aid of anesthesia is beyond comprehension. Although cataract extractions and other operations on the eye have been performed with ether and with chloroform anesthesia for the past eighty-seven years, the undesirable results incident to the use of these drugs, particularly in cataract extractions, are obvious.

The era of modern ophthalmic surgery really began in 1884, with the introduction of cocaine by Koller, which permitted operations to be performed on the eye of the conscious patient with comparatively little pain. So it is during the past fifty-nine years that ophthalmic surgery has made such tremendous strides, due, in great measure, to the ability to produce local anesthesia.

Although the general effect of chewing coca leaves had been known for centuries, the alkaloid cocaine was unknown until 1855, when it was discovered by Gaedcke,[1] who called it erythroxylin. The anesthetic properties of the drug were not made use of until Koller [2] made his memorable presentation in 1884, nearly thirty years later. After this the use of cocaine as a local anesthetic was recognized and quickly gained worldwide favor.

1. Gaedcke: Arch. d. Pharm. **121**:141, 1855.
2. Koller, C.: Ber. ü. d. ophth. Gesellsch. **16**:60, 1884.

In prehistoric times the coca plant was cultivated in Peru and Bolivia. Moodie [3] pictured a prehistoric surgical operation in Peru. The surgeon is said to have in his cheek a quid of coca leaves, which he applies to the wound to ease the patient's distress. According to Braun (page 71),[4] Scherzer was the first to bring a large quantity of coca leaves to Europe. Later, Wöhler, of Göttingen, Germany, received some of the leaves, from which his pupil Niemann [5] isolated the powerful alkaloid and named it cocaine. The first reports in regard to the anesthetic properties of cocaine were made by Scherzer,[6] who noticed that the chewing of coca leaves caused a feeling of numbness of the tongue; this was also observed by Niemann. In 1879 von Anrep [7] noted the anesthetic action of cocaine on the skin after the injection of a weak solution but observed only the mydriatic action of the drug when it was instilled into the conjunctival sac. In 1880 Coupard and Bordereau [8] observed in animals the loss of the corneal reflex after the use of solutions of cocaine.

Koller's report of his observations, four years later, seems to mark the real introduction of cocaine as an anesthetic agent. Although other investigators have claimed priority in the use of cocaine as an anesthetic, Koller is usually given the credit, for, as Darwin [9] said: "In science the credit goes to the man who convinces the world, not to the man to whom the idea first comes." This new anesthetic was favorably received, and during the next two or three months after Koller's communication, in Heidelberg, Germany, on Sept. 15, 1884, many reports of its use appeared in the literature.

In December 1884, Knapp [10] published a complete summary of the literature in regard to the use of cocaine in all branches of surgery. In this he credited Bull, of New York, with being, probably the first surgeon in America to employ cocaine for induction of anesthesia (Oct. 8, 1884). Reports of enucleations with the use of cocaine by Knapp, Cocks and Turnbull are also included in the article. The simple and effective method of a retrobulbar injection which was used by Knapp to produce anesthesia of the globe for enucleation deserves particular recognition. Superficial anesthesia was produced by the instillation of a 4 per cent solution of cocaine hydrochloride. By means of fixation forceps the globe was then forcibly drawn toward the nose; a needle was introduced as far as the posterior pole of the globe, and 6 minims (0.372 cc.) of a 4 per cent solution of cocaine hydrochloride was injected. The eye was enucleated without pain.

Cocks instilled a 4 per cent solution of cocaine hydrochloride every three or four minutes during the operation. After raising the conjunctiva, he injected a 4 per cent solution of cocaine hydrochloride along the tendon of each rectus muscle and made another injection in the orbit behind the globe.

Turnbull employed cocaine by instillation only. After opening Tenon's capsule, he instilled cocaine before each tendon was severed. The scissors to cut the optic nerve were then introduced, and, after thorough sponging, several drops of a 4 per cent solution of cocaine hydrochloride were allowed to flow down the blades. After a wait of one minute the nerve was severed.

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3. Moodie, Ray Lee: Studies in Paleopathology, Ann. M. Hist. **5**:374, 1923, frontispiece.
4. Braun, H. F. W.: Local Anesthesia, Philadelphia, Lea & Febiger, 1924.
5. Niemann, A.: Ueber eine neue organische Base in den Cocablättern, Inaug. Dissert., Göttingen, E. A. Huth, 1860.
6. Scherzer: Ueber die pesuanische Coca, Stuttgart, 1860.
7. von Anrep, B.: Arch. f. d. ges. Physiol. **21**:38, 1870.
8. Coupard and Bordereau: Compt. rend. Soc. de biol., 1884, p. 1.
9. Darwin, F.: Eugenics Rev. **6**:1, 1914-1915.
10. Knapp, H.: Arch. Ophth. **13**:402, 1884.

These operations are mentioned particularly because of the historical interest they possess. However, enucleation with local anesthesia did not meet with general approval, probably owing to the alarming toxic effects that followed the strong solutions of cocaine used.

Some years later enucleation with local anesthesia was revived by Siegrist,[11] Löwenstein,[12] Seidel[13] and others, a weaker solution of cocaine, procaine or a similar substance being used. At present enucleations are done easily and painlessly with the use of local anesthesia, and with many surgeons it is the procedure of choice.

Infiltration with cocaine near or into the nerves was first reported in 1884 by Hall,[14] who observed that the tissues supplied by the nerves so treated became anesthetic. The technic of this procedure, which is now spoken of as nerve block, has been greatly improved and is in general use.

The toxicity of cocaine was quickly realized, but not until many unhappy results had followed its use, particularly in general surgery, in which larger quantities were used than in ophthalmic surgery. Many of the unfortunate accidents were probably due to the rapid injection and absorption of strong solutions of the drug.

Also, epinephrine had not yet been discovered. Cocaine still holds an important position in ophthalmic surgery, although in many procedures it has been replaced by less toxic and more efficient drugs.

After the discovery of the chemical composition of cocaine and its atomic grouping by Einhorn,[15] the synthetic preparation of this alkaloid became possible. This served as a starting point for interesting experiments in which the anesthesiophore atomic group was combined with new atomic groups. This chemical research resulted in the discovery of a number of new local anesthetics, such as phenacaine, eucaine and compounds of the orthoform group, and later of stovaine, amydricaine, and procaine—certainly a triumph of an exact science.

In 1901 Takamine[16] isolated the active salts of the adrenal gland and added epinephrine to the pharmacopeia. This contribution increased greatly the efficacy of local anesthesia and facilitated operative procedures in which considerable bleeding is encountered.

Until the introduction of ether, in 1846, the success of operations on the eye was largely dependent on the speed of the surgeon and the stoicism of the patient. Although the necessity for speed has long since been eliminated by the improved methods of both general and local anesthesia, some ophthalmic surgeons still extol the virtue of speed and place unwarranted emphasis and importance on it.

Such drugs as cocaine, ether and chloroform were known for years before their anesthetic properties were fully appreciated and used. So, also, much of the present knowledge of physiology and anatomy is allowed to lie dormant, and in the technic of local anesthesia many of the advances and improvements are not given the recognition that they deserve.

SENSORY DISTRIBUTION TO REGION OF THE ORBIT

Since the aim of anesthesia is to eliminate pain, a consideration of the distribution of pain sense in the different tissues with which the ophthalmologist has to deal seems a logical approach to the problem. No doubt the pain sense varies in different persons, as does the interpretation of pain by different surgeons. The literature

11. Siegrist, A.: Klin. Monatsbl. f. Augenh. **45**:106, 1907.
12. Löwenstein, A.: Klin. Monatsbl. f. Augenh. **46**:592, 1908.
13. Seidel, E.: Klin. Monatsbl. f. Augenh. **49**:329, 1911.
14. Hall, R. J.: New York M. J. **40**:643, 1884.
15. Einhorn: München. med. Wchnschr. **46**:218, 1899.
16. Takamine: Therap. Gaz. **17**:221, 1901.

furnishes many examples of this, and the following case illustrates the point: An operation for the removal of a toe nail was reported by Shaw,[17] who stated that after the toe was wrapped in cotton saturated with cocaine for a few minutes, the nail was divided and removed without pain.

According to Braun,[18] the skin, with its innumerable nerve endings, is probably the most sensitive tissue in the body. Although the skin has a highly developed pain and tactile sense, which is more acute in some areas than in others, it can hardly be considered more sensitive than the cornea. One can easily demonstrate this by touching the skin and then the cornea with a wisp of cotton. However, the skin is extremely sensitive to pain, and if the surgeon is to retain the confidence of the patient, it is well for him to avoid causing unnecessary pain. In the introduction of the anesthetic agent, the pain caused when the skin is pierced may be practically eliminated if the skin is rendered insensitive by the production, first, of an intradermal wheal.

The loose subcutaneous tissue possesses little, if any, sensibility, although numerous conducting nerves containing sensory fibers for the skin traverse this area. One may produce pain by cutting or by making traction on these nerves, but, as a rule, no pain will be experienced when a needle passes through the subcutaneous tissue unless it comes in contact with one of the small sensory nerve trunks.

In this respect, the orbital tissue corresponds to the subcutaneous tissue, and the introduction of a needle, as in orbital injections, is practically painless if the skin has first been anesthetized by the production of a small intradermal wheal, provided the periosteum is not pricked with the needle and the muscles and nerves are avoided.

The nerve distribution in muscles is practically the same as that in connective tissue, so that very sharp pain is caused if the sensory nerves are touched. However, if the nerve is not encountered, a needle may be introduced into healthy muscle tissue without its causing pain. Muscle fascia and the surrounding connective tissue are distinctly sensitive, so that if the needle is passed through these tissues without the parts being first anesthetized pain is experienced.

Tendon tissue appears to be without sensibility. However, the connective tissue covering the tendon and the tendon sheaths possesses a varying degree of pain sense. Joint capsules, ligaments, synovial membranes and periosteum are extremely sensitive, and if they are pricked by the needle considerable pain may be produced. There seems to be some difference of opinion with regard to the sensitivity of the bones, but in general they can be considered as having little pain sense. This is not true of the medulla of long bones, in which, however, sensibility is not acute. Cartilage is insensitive, but the perichondrium is well supplied with nerves and is very sensitive to pain.

Since the margins of the eyelids are richly supplied with sensory nerves, they are the most sensitive part of the lids. Although the conjunctiva and the cornea are extremely sensitive, they can easily be rendered insensitive by the instillation of a few drops of an anesthetic agent if the eye is not red and inflamed.

MENTAL ATTITUDE OF THE PATIENT AND THE SURGEON

In operations performed with local anesthesia, particularly ophthalmic procedures, the proper mental attitude of both the patient and the surgeon is so

17. Shaw: New York M. J. **40**:588, 1884.

18. Braun,[4] p. 29.

important that a consideration of this subject seems warranted. Often the entire important procedure of the production of anesthesia is consigned to the assistant or to the intern, and quite properly too, provided he has been instructed adequately and is sufficiently familiar with the technic. In visiting large clinics one occasionally sees a patient who is so frightened by the preliminary preparation that the surgeon finds it difficult to quiet him and to regain the confidence necessary to obtain his full cooperation.

Apprehension is usually the greatest cause of suffering before the operation. In order to assure the patient that every precaution will be taken to insure his safety and comfort, it is necessary to inspire confidence, and the surgeon who can obtain this rarely encounters a patient who "behaves badly." The patient is vitally interested in having a successful result; so if he does not cooperate, it is frequently the fault of the surgeon. Certainly, in most instances, the surgeon begins with the advantage of having the patient's confidence; otherwise he would not have entrusted his most valuable possession, sight, to that surgeon. The operator should strive to strengthen this confidence and endeavor in every way possible to avoid undermining it. The aim of all surgeons is to secure good results. With operators of equal surgical skill, the one who gains the patient's confidence will undoubtedly have the greatest number of good results.

Local anesthesia demands and allows deliberation and care in all the manipulations of the operator. A confident, orderly atmosphere in the hospital, particularly in the operating room, and a comfortable, well padded operating table invite complacency. In the preparation of the patient, careful attention should be given to the minor details. Patients are particularly sensitive at this time, so that the instillation of a drop without warning or the accidental introduction of soap, iodine, or the like, in the eye greatly upsets the patient's equilibrium. Hurried or inefficient attempts to produce anesthesia may inflict unnecessary pain. Even the slightest needle prick will, in certain patients, cause considerable pain, for at the time the anesthetic is introduced the patient is most apprehensive, his sensitivity to pain is greatly exaggerated and each succeeding needle prick seems to be of increasing intensity. It is, therefore, of the greatest importance to induce anesthesia painlessly in order to retain and strengthen the patient's confidence.

Haste should be avoided, and the operation should not be attempted until complete anesthesia has been secured. Any complaint of pain usually indicates the inefficient administration of the anesthetic rather than a defect in the anesthetic procedure, the anesthetic agent or the patient. It is best not to proceed with the operation until complete anesthesia is obtained, and then it will rarely be necessary to resort to the well known "vocal anesthesia." For the surgeon to scold and blame the patient, the assistants or the nurses or to charge the failure to faulty instruments indicates usually a lack of self control on his part.

With a limited number of patients the temperament or mental makeup offers difficulties. Patients undoubtedly differ to as great an extent in their mental as in their physical characteristics; so they must be approached as differently as one would approach the various surgical conditions encountered.

In many cases fear is the chief barrier that prevents one from operating on children with the use of local anesthesia. An experienced nurse who has won the confidence of a child can often engage its attention with interesting conversation, which allows the operation to proceed smoothly. Often with adults, if the attention of the patient can be diverted, the operation will be easier and pleasanter for all concerned. Remarks such as "Did that hurt?" or "We are almost finished" should certainly be avoided.

While the use of a sedative or hypnotic, such as one of the barbituric acid group, before operation aids greatly in giving one a confident, quiet and complacent patient, it is, nevertheless, of secondary importance and should be considered only as an aid. Sedatives of the barbituric acid group have the added value of counteracting to some extent the toxic action of the anesthetic agent.

AGENTS FOR LOCAL ANESTHESIA

Since the advent of cocaine, chemists have produced numerous substitutes, some of which have in large measure replaced cocaine, particularly in cases in which the anesthetic is injected for block or infiltration anesthesia. A consideration of all the local anesthetic agents is obviously unnecessary. Only a few of the more generally accepted ones which are particularly suited to ophthalmic surgery will be considered.

Instillation Anesthesia.—For instillation anesthesia, cocaine, phenacaine (holocaine), butacaine (butyn) and tetracaine (pontocaine) are the agents most frequently used.

Cocaine: Cocaine hydrochloride is the salt of cocaine that is generally used as an anesthetic agent. It is a white, crystalline powder, readily soluble in water and in alcohol. According to the report of the Committee on Local Anesthetics in Ophthalmic Work of the American Medical Association,[19] cocaine may be sterilized by boiling without impairment of its efficacy. Braun [20] stated that no material loss of cocaine follows a single, rapid boiling of a small quantity of the solution, whereas repeated boilings of large quantities cause a diminution in the cocaine content, with diminished activity of the solution.

To prepare a fresh sterile solution of cocaine, Mikulicz recommended that the cocaine be dissolved in alcohol in a sterile flask stoppered with cotton and that when the alcohol evaporates sterile water or saline solution be added to the residue. According to the aforementioned report,[19] the addition of 5 grains (0.325 Gm.) of boric acid powder to each ounce (29.5 cc.) of solution tends to preserve the solution, so that it will remain active longer.

The physiologic action of cocaine is that of a protoplasmic poison. It paralyzes temporarily, and without permanent damage, the sensory and motor peripheral nerves, and if brought into direct contact with sensory or motor fibers, it causes them to lose their power to transmit impulses. The sensory fibers are affected more quickly and easily than the motor fibers.

It is generally agreed that for infiltration or block anesthesia there are safer and better drugs than cocaine. In referring to these types of anesthesia, Braun [21] dismissed cocaine as follows: "Cocain, at least in surgery, has become obsolete." Labat [22] stated (page 39) that "cocain has been practically discarded except for contact anesthesia of mucous membrane."

General cocaine poisoning will be considered only briefly, since such highly toxic drugs as cocaine are no longer accepted for infiltration or block anesthesia, and in the amounts used in ophthalmologic practice it would be unusual for toxic symptoms to develop from topical use of the drug. When toxic symptoms do appear, the chief disturbance occurs in the central nervous system, which is most sensitive to cocaine. In mild forms of poisoning, appearing shortly after the use of cocaine, there is a sudden, usually transient, attack of vertigo, which may become more severe, particularly if it is allowed to continue. Fainting, a small compressible pulse, cold

19. Report of the Committee on Local Anesthetics in Ophthalmic Work of the American Medical Association, Tr. Sect. Ophth. A. M. A., 1921, pp. 297-298.

20. Braun,[4] p. 93.

21. Braun,[4] p. 184.

22. Labat, G.: Regional Anesthesia, Philadelphia, W. B. Saunders Company, 1928.

sweat, irregular and difficult respiration, formication and cold extremities may be followed by loss of consciousness. The pupils become dilated and fixed. Vomiting is frequent. Severe poisoning usually begins with epileptiform convulsions, exophthalmos and loss of consciousness; death results from paralysis of the respiratory center.

The local application of cocaine causes contraction of the small capillaries and arteries, especially of the mucous membrane, resulting in localized ischemia, followed by congestion or dilatation of the vessels. While the newer agents for local anesthesia have an anesthetic action similar to that of cocaine, they do not cause ischemia.

For superficial or surface anesthesia, one instillation of a 4 per cent solution of cocaine hydrochloride produces adequate anesthesia in five minutes. For deep anesthesia, such as is required for cataract extractions, four instillations at three minute intervals is the procedure usually followed, and it produces the maximum effect in about fifteen minutes.

In addition to the anesthetic action on the eye, cocaine dilates the pupil and causes slight paralysis of accommodation. The corneal epithelium is also affected, becoming desiccated, particularly if the eyelids are not kept closed or the cornea is not kept wet with a solution of boric acid or some similar solution. The tendency of the patient to keep the eyelids open and not to wink after the use of cocaine increases the possibility of the occurrence of this condition. Cocaine also devitalizes the cornea, so that if the drug is used for cataract extractions, collapse of the cornea and delay in closure of the corneal wound are more frequently observed than with such anesthetic agents as butacaine and tetracaine. For this reason, cocaine is not so desirable for the operation of corneal transplantation, and its use may account for some of the grafts' becoming cloudy.

Cocaine hydrochloride is used in from 1 to 10 per cent solutions, but the 4 per cent solution is generally preferred. In operations on muscles some surgeons use powdered cocaine over the operative field. Except when its use is contraindicated, epinephrine is instilled in conjunction with the anesthetic agent, in order to constrict the vessels, decrease bleeding and prolong the anesthetic effect. Sollmann [23] stated that for surface anesthesia the addition of an equal volume of a 0.5 per cent solution of sodium bicarbonate to a solution of cocaine increases the activity of the cocaine one to two times.

Phenacaine: Phenacaine was prepared in 1897 by Taeuber by combining molecular quantities of acetophenetidin and phenetidin (ethyl ester of paraaminophenol). Although the basic compounds of phenacaine are insoluble in water, the hydrochloride of the drug is soluble up to 2.5 per cent and is the salt used. Phenacaine hydrochloride is incompatible with alkalis and their carbonate bases, so that porcelain instead of glass dishes should be used in its preparation. It is considerably more toxic than cocaine and should never be used hypodermically. On instillation it produces considerable irritation, smarting and congestion of the conjunctiva. The anesthetic effect is produced more quickly than that of cocaine. There is a slight antiseptic action. Phenacaine does not dilate the pupil, does not cause desiccation of the cornea and does not devitalize the cornea, as cocaine does.

The solution of phenacaine hydrochloride may be sterilized by boiling without its efficacy being affected. A 1 per cent solution has been widely used to produce surface anesthesia for tonometry and the removal of foreign bodies. It has also been employed by some surgeons for intraocular operations, such as extraction of cataract, and, with the use of epinephrine to produce ischemia, it is a satisfactory

23. Sollmann, T.: The Comparative Efficiency of Local Anesthetics, J. A. M. A. **70**:216 (Jan. 26) 1918.

topical anesthetic. However, phenacaine has been pretty generally replaced by butacaine, which, in turn, is now being challenged by tetracaine, because the latter causes little smarting when it is instilled in the conjunctival sac.

Butacaine: Butacaine sulfate was introduced in 1920; it is a white, amorphous powder, prepared synthetically by Adams, Kamm and Volwiler. It is freely soluble in water and may be sterilized by boiling without impairment of its efficacy. It does not deteriorate rapidly on exposure to light or air, possibly because it possesses certain antiseptic properties. Butacaine causes precipitation of sodium and other chlorides; so it should not be dissolved in a saline solution.

For instillation butacaine sulfate is generally used in a 2 per cent solution. After a single instillation of a 2 per cent solution, surface anesthesia is usually produced in one minute and lasts from fifteen to thirty minutes. However, two instillations are preferable, an interval of two or three minutes being allowed between each instillation. Patients are not equally susceptible to the action of butacaine, but with two instillations sufficient anesthesia is produced in practically all persons to permit the removal of rather deeply embedded foreign bodies without pain. The depth, degree and duration of the anesthesia are increased by further instillations. Four instillations of a 2 per cent solution of butacaine sulfate at intervals of three minutes produce profound anesthesia of the cornea and conjunctiva, and in five or ten minutes after the last instillation the maximum effect is reached. The average duration of anesthesia is about thirty minutes, but the effect may last as long as an hour. The first instillation causes considerable smarting and irritation and slight hyperemia of the conjunctiva, which may be controlled with epinephrine.

In the literature no toxic symptoms have been reported to follow the topical use of butacaine in the conjunctival sac. The Research Committee of the Council on Pharmacy and Chemistry of the American Medical Association stated that, when injected hypodermically into albino rats, butacaine was two and one-half times as toxic as cocaine.

In the report of the Committee on Local Anesthetics of the Section on Ophthalmology of the American Medical Association,[24] butacaine was compared with cocaine as a local anesthetic, and the unanimous opinion expressed by the Committee was that for surface anesthesia butacaine is superior to cocaine, as it acts more quickly and is accompanied by fewer complications. There are no objectionable effects, such as dilatation of the pupil or desiccation of the cornea, and the anesthesia with a 2 per cent solution of butacaine sulfate is more profound than that with a 4 per cent solution of cocaine hydrochloride. Also, no toxic symptoms were noted after the instillation of a 2 per cent solution of butacaine sulfate in the conjunctival sac.

Tetracaine: Tetracaine, a derivative of procaine, is one of the newer local anesthetics. For instillation anesthesia its chief advantage over phenacaine and butacaine is the fact that it causes little smarting. It is an odorless, white, crystalline substance, readily soluble in water and fairly stable when exposed to light and air. It may be sterilized by boiling without decomposition and without impairment of its efficacy. According to Wilmer and Paton,[25] it is compatible with epinephrine, atropine, homatropine, boric acid, pilocarpine, resorcinol, scopolamine and zinc sulfate.

Various reports agree that when it is given subcutaneously the toxicity of tetracaine is ten times as great as that of procaine, but as it is effective in one-tenth the

24. Bulson, A. E., Jr.: Butyn, a New Synthetic Local Anesthetic: Report Concerning the Clinical Use, J. A. M. A. **78**:343 (Feb. 4) 1922.

25. Wilmer, W. H., and Paton, R. T.: Tr. Am. Ophth. Soc. **30**:31, 1932.

strength of the effective dose of procaine, the relative toxicity is therefore about the same as that of procaine.

Schmidt,[26] Lundy and Essex,[27] Singer,[28] and Kiess [29] reported on the topical use of tetracaine for infiltration and for spinal anesthesia in over 10,000 cases, representing a great variety of operations. They did not report having observed any toxic effects. Schülein [30] recommended tetracaine because of the prolonged action and the low cost of the drug. He stated that with tetracaine the duration of anesthesia is from four to six hours, whereas with procaine it is only from one and a half to two hours.

Because tetracaine has a powerful hypotonic action in aqueous solution, Wilmer and Paton [25] advised the use of isotonic solution of sodium chloride as a solvent. These investigators, in reporting the use of tetracaine in over 500 cases at the Wilmer Ophthalmological Institute, stated that it possesses definite advantages over other local anesthetics.

After two instillations of tetracaine in 0.5 per cent solution, surface anesthesia is produced in about one minute and lasts about twenty minutes. With more instillations at two to three minute intervals deeper and more prolonged anesthesia is obtained. There is some lacrimation, but practically no hyperemia. The corneal epithelium is not disturbed, and the size of the pupil, accommodation and tension are not affected.

When these four local anesthetic agents, cocaine, phenacaine, butacaine and tetracaine, are compared, it will be noted that the chief advantages of tetracaine are as follows:

1. Little smarting or congestion of the conjunctiva is produced when the drug is instilled in the eye.

2. The action is rapid and prolonged.

3. Tetracaine does not dilate the pupil or devitalize the cornea.

4. Tetracaine has the same relative toxicity as procaine and therefore a great deal less than cocaine, phenacaine and butacaine.

Occasionally tetracaine may produce conjunctivitis and dermatitis of the lids, similar to that with atropine and other drugs commonly used in the eye. However, this reaction is rarely observed with tetracaine.

Comment: While cocaine is still popular, the chief objections to its use are that it dilates the pupil and may influence the tension. Also, cocaine causes desiccation of the corneal epithelium and tends to devitalize the cornea. The deleterious action of cocaine may retard healing, and in keratoplastic operations it may be responsible for some of the grafts becoming cloudy. It also may account for the more frequent collapse of the cornea observed in cases of cataract extraction. Phenacaine and butacaine have practically no advantages over tetracaine, and they cause more smarting when instilled in the eye. Metycaine, and possibly other local anesthetic agents, may compare favorably with tetracaine, but tetracaine has been found very satisfactory.

Superficial anesthesia sufficient for the removal of foreign bodies from the cornea, tonometry and such minor procedures can be produced in from one to five minutes, the period depending on the anesthetic agent used. Two instillations should be

26. Schmidt, H.: Chirurg. **3**:97, 1931.
27. Lundy, J. S., and Essex, H. E.: Proc. Staff Meet., Mayo Clin. **6**:376, 1931.
28. Singer, R.: Wien. med. Wchnschr. **81**:1593, 1931.
29. Kiess, T.: Zentralbl. f. Chir. **57**:3090, 1930.
30. Schülein, M.: München. med. Wchnschr. **78**:1475, 1931.

made. The patient is directed to look upward; the lower lid is drawn down, and several drops of the anesthetic are instilled in the lower cul-de-sac. In two or three minutes the second instillation is made. The patient is asked to look downward; the upper lid is raised, and several drops of the anesthetic are allowed to flow under the upper lid and down over the cornea. In this way a more complete anesthesia is produced than if both instillations were given in the lower cul-de-sac.

With two instillations tonometry is less likely to be unpleasant, a point of some importance, since the patient can more easily hold his eye quiet. Also, if the eye is not well anesthetized, the patient may conclude that if this apparently minor procedure is painful, an operation would be unbearable. For deeper anesthesia, four instillations at three minute intervals should be made, and in fifteen minutes the maximum effect is usually produced. To produce ischemia and obtain a bloodless field, as well as to prolong the anesthesia, several drops of epinephrine hydrochloride, 1:1,000, are also instilled.

Regional or Infiltration Anesthesia.—Procaine: In selection of the anesthetic for regional or infiltration anesthesia, the first consideration should be that of safety. Second, adequate anesthesia should be produced quickly, without injury or irritation to the tissues, and should be of sufficient duration to allow the surgeon ample time to complete the operation. Third, the anesthetic agent should be easily available; its preparation should be simple, and sterilization by boiling should not cause deterioration.

Procaine fulfils these requirements better than any local anesthetic yet produced. Braun [31] stated that the toxic action of procaine is less pronounced than that of any hitherto known anesthetic and that it possesses scarcely any irritating properties. Labat [32] said: "Novocain [procaine] has stood the test for many years and is the drug of choice." The Research Committee of the Council on Pharmacy and Chemistry of the American Medical Association reported that procaine is the least toxic local anesthetic. The Committee on Local Anesthetics in Ophthalmic Work of the American Medical Association [32a] recommended it as the most desirable drug for infiltration anesthesia. Procaine is now so generally accepted as the safest and best agent for production of all regional and infiltration anesthesia that a consideration of other more toxic and less satisfactory drugs seems unnecessary.

The salt (hydrochloride) of procaine crystallizes from alcohol in the form of needles, which melt at a temperature of 156 C. It is soluble in equal quantities of water, producing a solution which is neutral in reaction and readily soluble in isotonic solution of sodium chloride. It is precipitated by alkaline reagents, such as sodium carbonate, to form an insoluble base. The crystals may be sterilized in the autoclave at a temperature of 110 C., and the watery solution may be sterilized without deterioration by boiling. Procaine is rapidly and completely absorbed locally, with no destruction of the tissues. As there is no peripheral effect on the blood vessels, the anesthetic effect is of short duration, so that it is necessary to use epinephrine to prevent the rapid absorption of the drug. With the addition of a small amount of epinephrine to the procaine, analgesia begins almost immediately after the injection and lasts two to three hours. Surgical anesthesia starts ordinarily in from five to ten minutes and continues one and one-half to two hours.

Procaine is not so well suited to production of anesthesia of mucous membranes by topical application, probably owing to the fact that it does not completely penetrate these membranes.

31. Braun,[4] p. 117.
32. Labat,[22] p. 40.
32a. Report of Committee on Local Anesthetics in Ophthalmic Work of the American Medical Association, Tr. Sect. Ophth., A. M. A., 1921, p. 308.

Braun [33] stated that a 0.4 per cent solution of potassium sulfate added to the solution of procaine hydrochloride greatly increases its anesthetic properties and in this concentration produces neither local nor general injury. When procaine is used in weak solutions and in doses sufficient to produce surgical anesthesia, its toxicity is negligible. Labat [34] gave the maximum dose of procaine hydrochloride for robust patients in good physical condition as 150 cc. of a 1 per cent solution and 60 cc. of a 2 per cent solution and emphasized the importance of the proper technic and of slow injection.

Although toxic symptoms are rarely encountered in ophthalmic operations, in which small quantities of the drug are used, a small amount of a 1 or 2 per cent solution injected rapidly or directly into the circulation may produce toxic symptoms. These symptoms are rapid pulse; palpitation; frequent, labored breathing; pallor of the face; cyanosis of the fingers, lips and ears; nausea; vomiting; cold sweats, and blurred vision. The rapid pulse, palpitation and rapid and labored breathing may also be due to epinephrine, are of short duration, lasting two to three minutes, and are not serious. If they do occur, the injection should be stopped; in the event of more serious symptoms, such as pallor, cyanosis and nausea, Labat [35] recommended subcutaneous injection of the following cardiac stimulant: caffeine, 0.25 Gm.; sparteine sulfate, 0.05 Gm.; sodium benzoate, 0.30 Gm., and strychnine sulfate, 0.001 Gm., put up in a 2 cc. ampule. After the patient has completely recovered, the injection of the procaine may be resumed.

It is important to prepare an isotonic solution, as hypertonic solutions cause shrinkage, and hypotonic solutions swelling, of the cells; with either, destruction of the tissue occurs. Isotonic solution of sodium chloride is consequently the solvent of choice.

Labat [36] emphasized the importance of the use of only freshly prepared solutions and of the addition, just before use, of 5 drops of colorless epinephrine hydrochloride, 1 : 1,000, to 100 cc. of the anesthetic solution, irrespective of the strength of the solution. Until 1920 Labat used 20 drops of epinephrine hydrochloride, 1 : 1,000, to every 100 cc. of the solution of procaine hydrochloride, but he gradually reduced the quantity to 5 drops and observed that toxic symptoms were less frequent and the reactions not so pronounced. Solutions of epinephrine that have become brown or pink should be discarded.

For the average patient the maximum dose of epinephrine hydrochloride, 1 : 1,000, given subcutaneously or intramuscularly is 10 minims (0.62 cc.). For elderly patients—those with arteriosclerosis and other lesions of the vascular system or diabetes—who are frequently encountered in ophthalmic practice, the amount should be reduced to 5 drops.

As the quantity of anesthetic solution used in ophthalmic operations is so small —rarely more than 10 cc.—one need be concerned little about exceeding the maximum safe dose of epinephrine if the proportion of 5 or 10 drops of the 1 : 1,000 dilution of epinephrine hydrochloride to 100 cc. of anesthetic solution is observed. The advantages usually ascribed to the addition of epinephrine to the procaine solution are that it hastens, intensifies and lengthens the anesthetic action of the procaine.

For minor operations that are performed in the office or the hospital, often requiring less than 1 cc. of 2 per cent procaine hydrochloride, a convenient and economical preparation is offered by several pharmaceutic houses. The procaine

33. Braun,[4] p. 119.
34. Labat,[22] p. 41.
35. Labat,[22] p. 45.
36. Labat,[22] p. 46.

and epinephrine are furnished in ampules containing 1 cc. of a 2 per cent solution of procaine hydrochloride with epinephrine hydrochloride 1 : 50,000 or 1 : 20,000 in concentration, sterile and ready for use. Larger ampules can also be procured, so that for the ophthalmic surgeon whose operations are somewhat scattered the use of such ampules, in which the dose is accurate and the percentage assured, is a decided advantage for both office and hospital work.

INSTRUMENTS

Syringes.—The Luer-Lok 2 and 5 cc. syringes (fig. 1) have been found satisfactory. These syringes have a lock which prevents the needle from coming off during the injection. They are lighter than the Labat syringes, which were formerly used, and are shown in figure 1. The Luer-Lok syringe is not damaged by being placed in a boiling sterilizer.

Needles.—Three sizes of needles are desirable: a 27 gage needle, 12.5 mm. in length, for subconjunctival injections, production of intradermal wheals and small subcutaneous injections; a 25 gage needle, 2 cm. in length, for deeper subcutaneous injections, production of akinesia, etc., and a 22 gage needle, 3.5 cm. long, for still deeper injections, such as retrobulbar injection within the muscular cone and blocking of the lid. Platinum-iridium needles seem to pass through the skin and tissues a little easier than steel needles. However, the rustless steel needles are satisfactory. The protective metal shield or holder for the needle prevents the point from becoming dulled or turned.

NERVE BLOCK, FIELD BLOCK AND INFILTRATION ANESTHESIA

Intradermal Wheal (fig. 2).—Before the skin is pierced for an injection, an intradermal wheal should be made to avoid production of unnecessary pain. For this purpose the 27 gage needle is used. The skin is stretched with the index finger, and the needle is held flat against the skin. The point of the needle is then introduced just under the epidermis, with the bevel edge up, and a little procaine-epinephrine solution is injected; this gives the site of the injection a somewhat bleached appearance, and the skin immediately becomes insensitive. For the skin of the lids, which is extremely thin and loose, a little fold may be made with the thumb and the index finger, the needle inserted at the summit of the fold and a similar wheal raised.

Local Anesthesia.—By this term, one usually refers to a procedure by which a part of the body is rendered insensitive to pain, whether by topical application of the anesthetic agent, infiltration at the site of the incision or regional anesthesia, which Labat [37] defined as "the result of a certain number of delicate surgical procedures by which it is possible to control pain temporarily by interrupting the sensory nerve conductivity of any region of the body. Motor function is occasionally interfered with."

Local anesthesia may be produced in two ways:

1. By nerve block, which is accomplished by injection of the anesthetic agent around the nerve supplying the part, so that the conductivity of the nerve is interfered with.

2. By field block, in which a wall of anesthesia is produced that renders the operative field insensitive to pain. This wall of anesthesia blocks all the nerves supplying the operative field and does not aim at any individual nerve, as in nerve block. First, an intradermal wheal is raised, through which the needle is introduced

37. Labat,[22] p. 1.

perpendicularly; the solution is injected slowly as the needle advances until it reaches the deepest layers of the soft tissues into which injection is to be made. The needle is then withdrawn until the point reaches the subcutaneous tissues, and the solution is slowly injected as the needle is withdrawn. The direction of the needle is then changed, the point being advanced along the line to be blocked and the needle introduced obliquely. This procedure is repeated several times, the needle being inclined more obliquely each time. A second wheal is then raised in line with the first, and the procedure is repeated until a wall of sufficient length is produced. It is often necessary to surround the operative area with a wall of anesthesia, the width depending on the nerve supply of the part to be operated on. This procedure is applicable in some plastic operations, and the principal advantage is the lack of infiltration of the operative field, which might interfere with healing or distort the tissues of the part involved. Also, if there is much scar tissue, the field cannot be infiltrated satisfactorily. With field block the anesthesia lasts longer because the anesthetic solution does not escape when the incision is made, as it does when the tissue along the site of the incision is infiltrated.

In production of regional anesthesia it is extremely important to visualize the anatomic features of the operative field, particularly with regard to the nerve

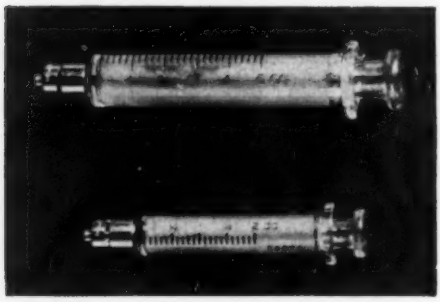

Figure 1

Figure 2

Fig. 1.—Luer-Lok syringes, of 2 and 5 cc. capacity.
Fig. 2.—Technic of production of intradermal wheal.

distribution and the bony landmarks that serve as a guide for the various injections. It is assumed that an accurate knowledge of the anatomic relations has been acquired, and the only references to the structure involved will be in connection with the technic employed.

Anesthetic Solution.—The anesthetic solution used in production of the following forms of anesthesia, unless otherwise stated, contains procaine hydrochloride, 2 per cent, and epinephrine hydrochloride, 1:1,000, 1 drop to each 5 cc. of anesthetic solution being used.

Infiltration Anesthesia.—After an intradermal wheal is raised, the needle is introduced through the wheal, and the anesthetic solution is injected slowly as the needle advances. If there are any large vessels in the vicinity, one should aspirate the contents of the needle occasionally to make sure that the needle has not entered a vessel.

Subconjunctival Injection.—For this procedure the 2 cc. syringe with the 27 gage needle is used. The lids are held open with the thumb and finger or with a speculum. The point of the needle is slid gently over the conjunctiva until a fold appears in front of the point; the syringe is then rotated, a boring effect being

produced. In this way even a dull needle passes easily and gently through the conjunctiva without the necessity of forceps or of quick jabbing.

Many excellent articles dealing with anesthesia in ophthalmic surgery have been published, but often specific and minute details regarding the technic have been omitted, the authors evidently assuming that their readers were familiar with them. From personal observation, this assumption seems hardly justified; it is with some temerity, however, that space is given to these important, although familiar, academic details.

RETROBULBAR INJECTION WITHIN THE MUSCULAR CONE

Indications.—Indications for this procedure are: (1) enucleation and evisceration; (2) intraocular operations, and (3) operations in which there is considerable traction on the muscles, such as advancement of an extraocular muscle.

Procedure and Comment.—The retrobulbar injection should be preceded by production of the usual instillation anesthesia. A small intradermal wheal is first raised a short distance below the inferior temporal margin of the orbit (fig. 2).

Figure 3

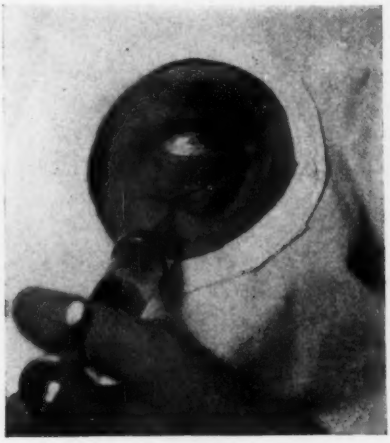

Figure 4

Fig. 3.—Skull, showing position of the needle for retrobulbar injection within the muscular cone.

Fig. 4.—Retrobulbar injection within the muscular cone (Atkinson, W. S.: Local Anesthesia in Ophthalmology, Tr. Am. Ophth. Soc. **32**:399, 1934).

When injection is to be made into the right orbit, the patient is directed to look upward and to the left (figs. 3, 4 and 5). The 22 gage needle, 3.5 cm. in length, is then introduced through the wheal, and the skin is moved upward with the needle so that the point just clears the inferior orbital margin. The needle is then directed upward and inward, midway between the external and the inferior rectus muscles, and is advanced toward the apex of the orbit for 2.5 to 3.5 cm., the distance depending on the size of the orbit. When the needle has reached a depth of from 2.5 to 3.5 cm., one should aspirate the contents before the solution is injected in order to be sure that the needle has not entered a vessel. However, in this location it is practically impossible to enter a vessel unless it is anomalous, since the vessels here are normally smaller than the needle and are freely movable. From 1 to 1.5 cc. of the 2 per cent solution of procaine hydrochloride is injected slowly, care being taken not to produce undue proptosis by too large an injection. For the majority of operations on the globe 1 cc. is sufficient, but for some operations on

muscles and for enucleation 3 cc. or more may be injected, as the proptosis does no harm and, particularly with painful eyes, more procaine is desirable.

Gentle massage with a rotary motion of the globe causes the procaine solution to become more thoroughly diffused within the muscular cone. In this way more complete and rapid anesthesia is produced. Anesthesia of the muscles is not obtained as rapidly as anesthesia of the globe; so for enucleation and operations in which there is considerable traction on the muscles it is advisable to wait five or ten minutes before beginning the operation.

By means of the injection within the muscular cone, all of the sensory nerves to the eyeball are blocked, and complete anesthesia of the globe is obtained regardless of how inflamed or painful the eye may be. Pressure on the inflamed eye, which before the injection caused severe pain, is not noticed now. The rectus muscles, except the superior rectus, are also rendered practically insensitive to traction.

The injection within the muscular cone also causes paresis of the rectus muscles, except the superior rectus, so that the patient cannot easily look down. There-

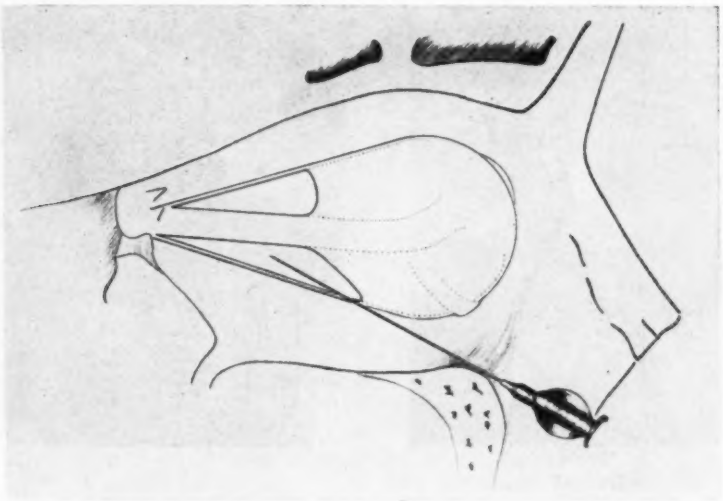

Fig. 5.—Diagrammatic sketch showing the eye turned up and away from the site of the injection, so that the fascial extension is moved forward and up, out of the way.

fore, for such operations as extraction of cataract it is advisable to use a suture under the superior rectus muscle (bridle suture), but with the rectus muscles inactive little traction is required to hold the eye in the desired position and better fixation of the globe is thus obtained. Also, with paresis of the rectus muscles the so-called squeeze, with loss of vitreous, caused by their contraction is less likely to occur.

The superior rectus muscle usually remains active and may be sensitive to traction. For prevention of pain when this muscle is picked up for the introduction of a bridle suture or for an operation in which there is traction on the muscle, additional anesthesia is necessary. This may be accomplished by an injection of 0.5 cc. of the anesthetic solution through the conjunctiva of the upper cul-de-sac. With the superior rectus muscle active and with paresis of the other extrinsic muscles, there is torsion of the globe, with rotation toward the nose. The torsion is increased when a suture is used under the superior rectus muscle. Therefore, if iridectomy is to be performed, it should be done from 10 to 15 degrees to the nasal

side, so that the coloboma will be at axis 90 when the muscles again function normally. While this is of no particular importance from a visual standpoint, a coloboma looks better in the vertical meridian.

When the eye is red and inflamed, as in cases of acute glaucoma, a subconjunctival injection should also be given, since the inflamed conjunctiva is not well anesthetized by topical application. Only the conjunctiva immediately surrounding the cornea for a distance of about 3 or 4 mm. is rendered insensitive by an injection within the muscular cone, since its nerve supply is from the ciliary nerves, which are within the cone. The rest of the conjunctiva is innervated by the supraorbital, supratrochlear, infratrochlear, infraorbital and lacrimal nerves, all of which are outside the muscular cone and are seldom reached by the anesthetic injected within the cone. When the eye is inflamed, less pain is caused if the subconjunctival injection is given after the retrobulbar injection. For the subconjunctival injection the

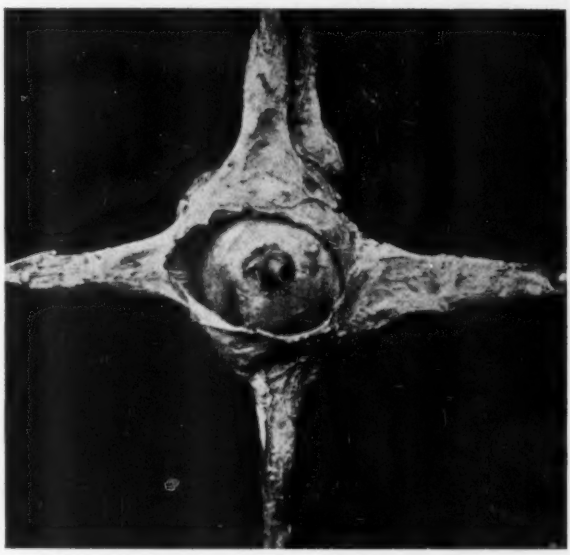

Fig. 6.—Posterior view of the eyeball, showing the fascial sheaths and, in part, the fascial extension which closes the spaces for a little distance back of the globe (from Whitnall [38]).

needle may be introduced through the insensitive conjunctiva directly surrounding the cornea, and if succeeding injections are made through blebs produced by the anesthetic, little pain will be experienced. In this way, pain due to the use of fixation forceps and to suture of the conjunctiva is avoided in eyes that are inflamed and painful.

With such complete anesthesia of the globe, and the eye practically immobile, together with paralysis of the orbicularis muscle, operations on the globe can be performed with the same deliberate precision that one can use when operating on a pig's eye.

If the patient looks upward and away from the site of the injection, access to the muscular cone is easier. The extension of the fascial sheath of the muscles which closes the spaces between the rectus muscles for a little distance back of the globe is in this way moved forward and upward, out of the way, so that the needle does not strike it, as is shown diagrammatically in figure 5. If the needle does strike the sheath, pain is produced. The sheath is also somewhat tough, and the needle

does not penetrate it easily, with the result that the globe rotates, which to some extent interferes with the introduction of the needle and the equilibrium of the patient.

Procaine injected within the muscular cone is apparently held within the cone, since the nerves outside the muscular cone are rarely affected. How is the solution of procaine held within the muscular cone, since there is no demonstrable fascia between the rectus muscles, posterior to the globe, as they converge to their attachment at the apex of the orbit? Anteriorly, the escape of fluid is prevented by the globe, surrounded by Tenon's capsule, and by the adjacent margins of the sheaths of the four rectus muscles, which are continuous with one another, with a wide sweep between them, as shown in figure 6 (from Whitnall,[38] fig. 15). In this manner, an intramuscular membrane is formed which, according to Whitnall, exists for a short distance behind the globe. Beyond this the muscles converge to their attachment through the annulus of Zinn, at the apex of the orbit, and their margins come closer together, so that comparatively little space is left between them. In this

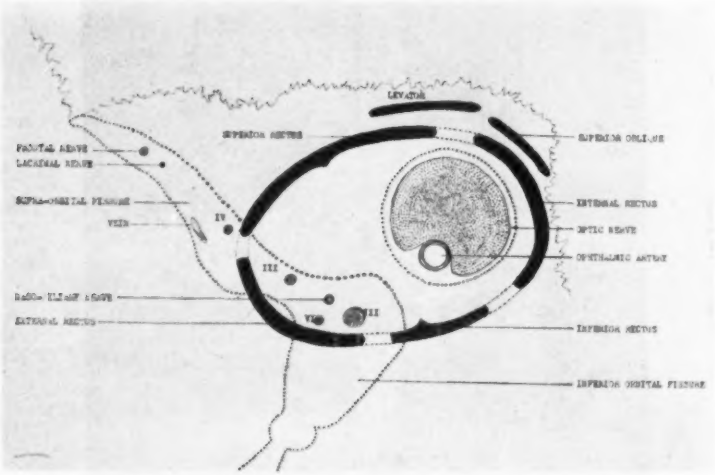

Fig. 7.—Scheme showing the position of the annulus of Zinn and the relative position of the nerves which enter the orbit through the superior orbital fissure, both within and outside the annulus (after Whitnall [38] and Poirier [Traité d'anatomie humaine, ed. 3, Paris, Masson & Cie, 1911, vol. 5]).

location the escape of the procaine solution is probably prevented, or at least retarded, by the closely packed layer of fat, with its connective tissue reticulum.

According to Whitnall,[39] the orbital fat which fills the entire space in the orbit not occupied by other structures varies in consistency in different regions. Within the muscular cone the fat has the appearance of large and loosely connected lobules, broken up by the passage of the nerves and vessels. Outside the muscular cone the fat is more firmly and closely packed and contains a quantity of connective tissue, which is closely attached to the muscular sheaths. No doubt this fat and connective tissue serves as a sufficient barrier so that the anesthetic solution is held within the cone, with production of anesthesia or blocking of the nerves that traverse it, namely, the nasociliary nerve, the long and short ciliary nerves, the ciliary ganglion, the optic nerve and all the motor cranial nerves except the trochlear, or fourth (figs. 7 and 8).

38. Whitnall, S. E.: The Anatomy of the Human Orbit and Accessory Organs of Vision, London, H. Frowde, Hodder & Stoughton, 1921.

39. Whitnall,[38] p. 297.

A decrease in the intraocular tension occurs after injection within the cone. If the amount of epinephrine hydrochloride (1:1,000) is increased to 3 drops to 5 cc. of the anesthetic solution, the decrease in the intraocular tension is greater, and a decrease of as much as 20 to 30 mm. of mercury may be noted in cases of acute glaucoma with high tension.

After the injection within the cone there is often noticeable deepening of the anterior chamber, particularly when the amount of epinephrine hydrochloride is increased. This indicates a decrease in the fluid contents of the eye posterior to the diaphragm. The incision is thus made easier in cases in which the chamber has been shallow. Also, with lowering of the pressure behind the lens the anterior capsule appears to become relaxed. In some cases the capsule of the lens, which prior to operation appeared to be too tight to be grasped with smooth forceps, was found to be loose after an injection within the cone, so that a good bite of the capsule could be obtained with the forceps.

With some methods of intracapsular extraction of cataract a decrease in the intraocular tension seems to be an advantage, as it lessens the possibility of prolapse

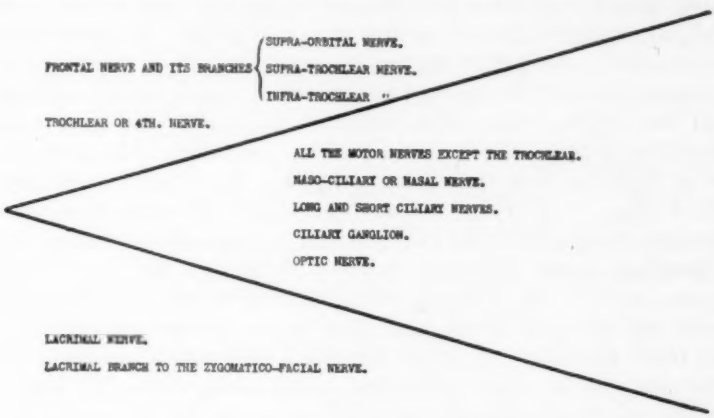

Fig. 8.—Diagram of nerves within and outside the muscular cone.

of the vitreous. In extraction with capsulotomy it is often more difficult to express the lens when the tension is decreased. With deeply set eyes the operation is somewhat facilitated if the eye is slightly proptosed. With lowered tension the iris may not prolapse well in corneoscleral trephinings, an incident which may be embarrassing. The decrease in the intraocular tension following the injection of procaine and epinephrine within the cone may be accounted for by the constricting action of the epinephrine on the arteries entering the globe, an effect which would tend to decrease the amount of blood entering the globe but would not impede its exit through the venae vorticosae, since the solution does not reach them, located as they are in the episcleral space. The free anastomosis of the venae vorticosae and other orbital veins with the facial vein allows free exit of the blood.

Newly formed aqueous, which Duke-Elder [40] termed "plasmoid aqueous," contains a larger proportion of colloid molecules, or plasma proteins, than does the normal aqueous. If this fluid is a dialysate due to dilatation of the capillaries of the ciliary body after escape of the normal aqueous, then injection of procaine and epinephrine within the cone not only would be of value as an anesthetic but, as

40. Duke-Elder, W. S.: The Nature of the Intra-Ocular Fluids, British Journal of Ophthalmology, Monograph Supplement 3, London, George Pulman and Sons, 1927.

pointed out by Friedenwald,[41] would prevent a postoperative increase in tension. The action of the epinephrine reduces the edema of the intraocular tissue, particularly that of the ciliary body and ciliary processes. Thus, dialysis is limited, and more nearly normal aqueous is formed, which diminishes the occurrence of so-called plastic iritis and the formation of synechiae.

This simple and efficient method of obtaining complete anesthesia of the globe has been revived, in a great measure, by the investigations of Löwenstein,[12] under the direction of Elschnig. With a view to injection into the ciliary ganglion for complete anesthesia of inflamed and painful eyes for enucleation, Löwenstein made a study of the exact location of this ganglion. By using a 5 cm. needle and introducing it through the conjunctiva temporally, just below the margin of the external rectus muscle, for a distance of 4.5 cm., he found the point to be just in front of the ganglion. Injections made in this manner within a few minutes produced complete insensibility of the most painful eyes.

Later, Elschnig modified the technic and introduced the needle through the skin at the inferior temporal margin of the orbit, rather than through the conjunctiva. The suffering caused by traction and pressure on painful eyes is thus avoided. The method also allows easier access to the muscular cone. In observations on the cadaver, Löwenstein noted that the optic nerve was often pierced with a 5 cm. needle and that a dye could be injected into the optic sheath readily without any dye's being found free in the orbit. This indicates how easily the optic nerve may be injured if a 4.5 or a 5 cm. needle is used. The procedure of Löwenstein has been referred to as injection into the ciliary ganglion. It is not surprising that many surgeons have hesitated to try it, and well they might, if the term is taken literally. It would, indeed, require the most exact technic to inject an anesthetic into the tiny, elongated ganglion, 2 mm. in length, lying as it does in the orbital fat within the muscular cone, about 1 cm. in front of the annulus of Zinn, between the external rectus muscle and the optic nerve, and close to the ophthalmic artery. That it is desirable to block the ciliary ganglion for many operative procedures is not questioned. But this can be done safely and easily by use of a 3.5 cm. needle and injection of the anesthetic solution more anteriorly within the muscular cone. In this location, because of the loosely connected fat lobules, the solution diffuses quickly and permeates thoroughly the ciliary ganglion, a fact undoubtedly appreciated by Knapp [10] nearly fifty years ago, when he employed and reported on the use of retrobular injection to produce anesthesia of the globe for enucleation.

Duverger,[42] after experiments on the cadaver with needles of various lengths, concluded that a 3.5 cm. needle is preferable for all orbital injections except those to be made at the extreme apex, and for these he found that the 4.5 cm. needle was just long enough to reach the optic foramen, but not to pass through it. With the 3.5 cm. needle Duverger was unable to pierce the optic nerve even after he had removed the temporal portion of the orbital wall so that he could observe the point of the needle, which had been introduced at the inferior temporal margin of the orbit. The point of the needle always eluded the nerve, because in this location the nerve is freely movable. The same property applies to the vessels here. They, also, are small, so that the risk of one's injuring them is slight. Nevertheless, the possibility of orbital hemorrhage is often mentioned as an objection to retrobulbar injection. Data on over 8,000 retrobulbar injections showed 8 orbital hemorrhages, 5 of which were admittedly due to faulty technic. In no instance was the eye damaged. If a

41. Friedenwald, J. S.: Am. J. Ophth. **15**:189, 1932.
42. Duverger, C.: L'anesthesia: Locale en ophthalmologie, Paris, Masson & Cie, 1920.

4.5 or 5 cm. needle is used and the point is carried to the apex of the orbit, obviously a vessel or the optic nerve might easily be pierced, because here they converge to reach their bony exits and are fixed. This danger is avoided by the use of a 3.5 cm. needle. Duverger also claimed that the best approach for injection within the muscular cone is from the inferior temporal margin of the orbit.

Branches and Sensory Distribution of the Ophthalmic and Maxillary Divisions of the Trigeminal Nerve

Nerve	Branches		Sensory Supply
Ophthalmic Division			
Frontal	Supraorbital	Internal frontal	Eyelids
		External frontal	Diploe and frontal sinus
	Supratrochlear	Branch to infratrochlear	Skin of forehead above head of eyebrow; Skin and conjunctiva of upper lid and side of nose
Nasociliary	Sensory root to ciliary ganglion; Two long ciliary nerves		Eyeball: Cornea; Iris; Ciliary body; Bulbar conjunctiva immediately around cornea; Sheath of optic nerve
	Infratrochlear, or external nasal		Internal canthus; Skin and conjunctiva mainly of upper lid, some of lower lid; Root of nose; Lacrimal sac; Canaliculi; Caruncle
	Internal nasal		Mucous membrane and anterior part of septum; Middle and inferior conchae; Lateral wall of nose; Skin and cartilaginous end of nose
Lacrimal			Lacrimal gland; Conjunctiva and skin of upper lid temporally
	Anastomosis with the zygomaticotemporal branch of zygomatic		Skin of forepart of forehead up to lateral side of orbit; Skin of cheek
Maxillary, or Second, Division			
Recurrent, or middle meningeal			Dura mater
Zygomatic	Temporal—communicates with lacrimal nerve		Skin of forepart of forehead up to lateral side of orbit
	Malar		Skin of cheek
Sphenopalatine			Palate and nasal cavity; Periorbita; Orbitalis muscle (involuntary); Sphenoid sinus; Posterior ethmoid cells; Minute fibers to: Lacrimal gland; Sixth nerve; Ciliary ganglion; Optic sheath
Posterior superior alveolar			Molar teeth; Gums; Mucous membrane of maxillary sinus
Middle superior alveolar			Two premolar teeth
Anterior superior alveolar			Lower part of lacrimal sac and nasal duct; Mucous membrane of nasal cavity; Incisor and canine teeth
Infraorbital	Inferior palpebral		Skin and conjunctiva of lower lid
	External nasal		Skin of side of nose
	Superior labial		Skin of cheek; Mucous membrane and skin of upper lip

Elschnig, in a personal communication, on Nov. 18, 1930, stated that he had made orbital injections regularly for more than five years. In about 2,000 intrabulbar operations, 4 orbital hemorrhages occurred, which Elschnig attributed to faulty technic. They did no harm, although in 1 case the operation had to be postponed until the following day.

The table shows at a glance the branches and the sensory distribution of the ophthalmic and the maxillary, or second, division of the trigeminal nerve.

OPERATION ON THE LACRIMAL SAC

Anesthesia for operation on the lacrimal sac is obtained by blocking the external nasal, or infratrochlear, and the infraorbital nerve.

The infratrochlear nerve is the direct continuation in the orbit of the nasociliary, or nasal, nerve after the larger branch leaves the orbit by passing through the anterior ethmoid foramen (fig. 9). The infratrochlear nerve lies close to the superior nasal wall of the orbit. It runs beneath the superior oblique muscle and the trochlea, and at this point there is a communicating branch to the supratrochlear nerve. Before piercing the septum orbitale, the infratrochlear nerve divides into several branches to supply the skin and the conjunctiva of the lids near the internal canthus, the canaliculi, the caruncle, the lacrimal sac and the root of the nose.

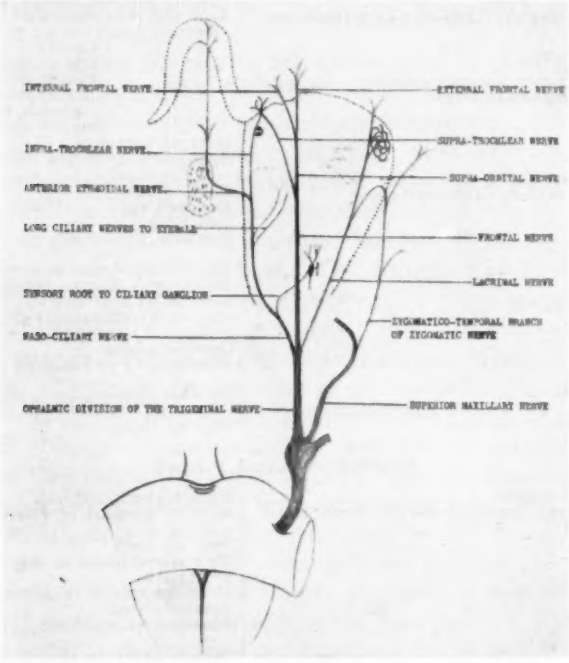

Fig. 9.—Diagram showing the distribution of branches of the ophthalmic division of the trigeminal nerve (after Whitnall, p. 342 [38]).

BLOCK OF THE INFRATROCHLEAR NERVE

A wheal is raised at the superior nasal angle of the orbit. A 3.5 cm. needle is then introduced through the wheal just a little above and to the nasal side of the trochlea (figs. 11 and 12). It should be kept close to the superior nasal wall, and a small quantity of procaine solution should be injected as the needle advances. At a depth of about 3 cm., 2 or 3 cc. of a 2 per cent solution of procaine hydrochloride with epinephrine hydrochloride is injected. On withdrawal of the needle, about 0.5 cc. should be injected along the orbital margin in the vicinity of the supraorbital notch anterior to the septum orbitale but close to the periosteum. Occasionally the angular vein is pierced, but if care is taken not to inject the solution while the needle is in the lumen, no harm will be done, although the resulting ecchymosis is annoying.

BLOCK OF THE INFRAORBITAL NERVE

Anatomic Relations.—The superior maxillary nerve enters the orbit through the inferior orbital fissure at about its midpoint and is then called the infraorbital nerve. The infraorbital nerve runs forward along the floor of the orbit, beneath the periorbita, in the infraorbital groove and canal. In the forepart of the infraorbital canal, a few millimeters from the foramen, the anterior superior alveolar branch is given off. This small branch passes through minute osseous canals to supply the lower part of the lacrimal sac and the nasal duct, as well as the incisor and the canine teeth. The infraorbital nerve passes through the infraorbital foramen and divides

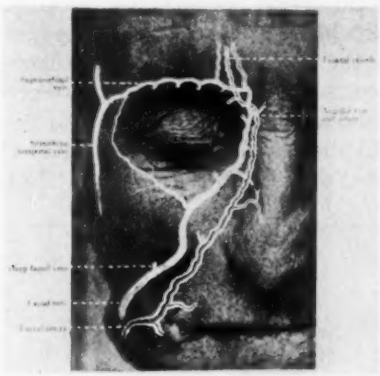

Fig. 10.—Approximate positions of the main vessels of the face which should be avoided, particularly in blocking the infratrochlear nerve (from Whitnall [38]).

Figure 11

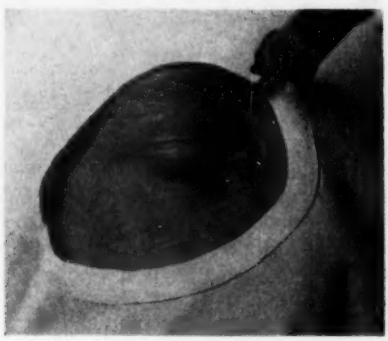

Figure 12

Fig. 11.—Skull, showing needle in position to produce block of the infratrochlear nerve.
Fig. 12.—Method of blocking the infratrochlear nerve.

into its terminal branches to supply the conjunctiva and skin of the lower lid, the skin of the cheek, the skin of the side of the nose and the skin and mucous membrane of the upper lip.

The infraorbital foramen is located about 0.5 to 1 cm. below the inferior orbital margin, about 2.5 cm. from the midline of the face, and in line with the supraorbital notch. Just below the center of the inferior orbital margin a depression can usually be felt in which the foramen is located.

The infraorbital canal is from 1 to 1.5 cm. in length, and the angle varies considerably in different skulls. A grooved depression leads up to the foramen and aids considerably in location of the latter. After the needle enters the foramen, the angle of the needle can be changed to correspond to the angle of the canal, and the needle can then be introduced easily, with practically no resistance.

Procedure.—A wheal is raised about 1 cm. from the middle of the ala of the nose. The same needle is introduced through the wheal almost to the bone and is then directed upward and temporally at an angle of about 20 degrees (figs. 13 and 14). As the needle advances the procaine solution is injected. The point of the needle is kept in contact with the bone until the foramen is felt; it is then introduced from 0.5 to 1 cm. along the infraorbital canal and 1 cc. of a 2 per cent solution of procaine hydrochloride is injected. The needle should be introduced into the canal; otherwise the anterior superior alveolar nerve will not be blocked. If this nerve is not blocked, some pain may be experienced when the sac is excised at the entrance of the nasal duct and when the nasal duct is curetted.

Labat [43] suggested that the left index finger be placed over the foramen, as an indication of its location and the axis of the infraorbital canal. This makes it easier

Figure 13

Figure 14

Fig. 13.—Skull showing the needle in position for injection into the infraorbital canal to block the infraorbital and the anterior superior alveolar nerve. (A 3.5 cm. needle was used in this picture in order better to show the angle, but for the injection the 2 cm. needle is used.)

Fig. 14.—Injection into the infraorbital canal to block the infraorbital and anterior superior alveolar nerves.

to find the foramen with the needle. Injection into this nerve produces anesthesia of the conjunctiva and the skin of the lower lid, the skin of the cheek, the skin of the side of the nose, the skin and the mucous membrane of the upper lip, the lower part of the lacrimal sac, the nasal duct and the incisor and canine teeth.

For the operation of dacryocystorhinostomy, besides block of the infratrochlear and the infraorbital nerve, topical anesthesia of the nasal mucous membrane should be obtained by the use of a nasal pack.

FIELD BLOCK FOR ANESTHESIA OF THE LIDS

Since there are so much overlapping and variation in the sensory nerve supply of the lids (fig. 15), it is simpler in operations involving a considerable area of the lids to produce a field block rather than to try to block the individual nerves.

43. Labat,[22] p. 117.

Procedure.—A wheal is raised at the center of the orbital margin (fig. 15), above or below, the position depending on which lid is to be blocked, and the 3.5 cm. needle is introduced through the wheal. The point of the needle should follow the orbital margin closely but should not penetrate the orbital septum. A 2 per cent solution of procaine hydrochloride with epinephrine hydrochloride (1:1,000) is injected slowly as the needle advances. In order to inject the anesthetic all along the margin and to infiltrate well the tissue around the nerves that emerge from the orbit, it is necessary to withdraw the needle a little and redirect it several times. The injection should not be made just under the skin, but should be done close to the periosteum. About 0.5 cc. of the anesthetic solution should be injected deep in the vicinity of the supraorbital notch.

After the anesthetic solution has been injected along one-half the superior or the inferior orbital margin, the location depending on which lid is to be blocked,

Fig. 15.—Right eye: The sensory nerve supply is shown by solid lines and the orbital margin by the dotted outline. Left eye: The dot and arrows indicate the site and direction of the injection (modified from Whitnall, pp. 20 and 176 [38]).

the needle is partially withdrawn; it is then redirected and the solution injected along the other half of the orbital margin.

For removal of small tumors of the lids, such as papilloma, infiltration at the site of the tumor is usually sufficient and is the simplest method. It also decreases the bleeding. For chalazion and operations involving the margin of the lid, which, owing to its rich nerve supply, is extremely sensitive, instillation anesthesia and infiltration at the site of the chalazion are often not sufficient to make the operation painless. However, instillation anesthesia and an injection of 1 cc. of the anesthetic solution through the conjunctiva of the cul-de-sac in the area corresponding to the chalazion more frequently produce complete anesthesia.

In operations in which large conjunctival flaps are raised, instillation anesthesia is often not sufficient and pain may be experienced, particularly when the flap is

sutured. This may be due to the incomplete anesthetization of the deeper layers of the conjunctiva or the subconjunctival tissue or to the rapid escape of the tissue fluids when the flap is raised, which favors the more rapid return of sensibility. This is also true in operations for pterygium. By the use of subconjunctival injections in such operations unnecessary pain is avoided.

AKINESIA

The desirability of production of temporary paralysis of the orbicularis muscle by block of the temporofacial division of the facial nerve for intraocular operations, particularly extraction of cataract, is so generally conceded that comment is unneces-

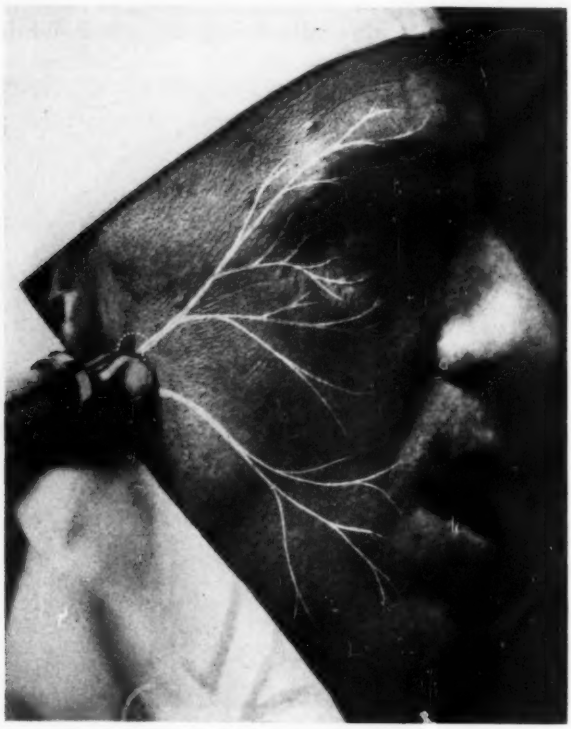

Fig. 16.—Method of block of the temporofacial division of the facial nerve to produce temporary paralysis of the orbicularis muscle.

sary. Of the various modifications of the van Lint method, the simplest and most satisfactory is the one proposed by O'Brien.[44] His description follows:

> The point of the injection is just anterior to the tragus of the ear, below the posterior portion of the zygomatic process and directly over the condyloid process of the mandible. Going straight inward with a sharp needle, one strikes the bony condyloid process at a depth of about 1 cm. As soon as this bone is felt with the needle, I begin injecting 2 per cent solution of procain hydrochloride, and, gradually withdrawing, inject about 2 cc. of solution. Lid paralysis begins to appear usually in from thirty to sixty seconds, and, after a very few minutes, is so marked that the patient is unable to close the lids, and the palpebral fissure is widely opened [fig. 16].

44. O'Brien, C. S.: Local Anesthesia in Ophthalmic Surgery, Tr. Sect. Ophth., A. M. A., 1927, p. 250.

The course of the branches of the seventh nerve which supply the orbicularis muscle often varies considerably, so that paralysis is not always secured with the first injection. If, after several minutes, the action of the muscle has not been affected, another injection should be given. The point of injection should be changed slightly, the more common variations in the course of the nerve being borne in mind. The object of the injection should be accomplished before one proceeds with the operation.

Sharp pain is caused if the periosteum of the condyloid process is pricked; it should therefore be avoided. After the injection of 2 to 3 cc. of the anesthetic solution, deep massage, with firm pressure over the site of the injection, will produce more rapid and complete paralysis of the orbicularis muscle.

When more prolonged paralysis of the orbicularis muscle is desired, as with patients who have a tic, with spasmodic contractions not only of the orbicularis but of other facial muscles, alcohol may be added to the solution injected.

The following solution causes little pain when injected, and the paresis lasts seven to ten days, sometimes longer: isotonic solution of sodium chloride U. S. P., 8 cc.; procaine hydrochloride, 0.2 Gm., and absolute alcohol, 2 cc. Less pain will be experienced if 1.5 cc. of the anesthetic solution is first injected and the syringe removed, the needle being left in place. Beginning paresis of the orbicularis muscle indicates that the needle is in the proper position. The syringe, containing the alcoholic solution, is then attached to the needle, and 1.5 cc. is injected.

In selection of the method of anesthesia for any operation, the age and temperament of the patient must be considered. With the use of instillation anesthesia only, many operations on the eye may be successfully performed with no apparent discomfort in a somewhat large proportion of patients. Because of this, the surgeon often does not make use of the procedures that produce more complete anesthesia, regarding them as unnecessary and superfluous. There is, however, a small percentage of patients for whom instillation anesthesia is not sufficient or who are less stoical than others. These are the patients who so often squeeze and damage their eyes seriously or who, through inability to hold the eye quiet, make the operation more difficult. Since it is not possible before operation to select the patients who will not squeeze the eyes or who will hold well, it seems wiser for the surgeon to err on the side of caution and always obtain complete anesthesia before he begins the operation.

For all operations on the globe in which a forcible contraction of the orbicularis muscle might injure the eye this muscle should be temporarily paralyzed. Also, in operations for strabismus the procedure is made easier if the lids are relaxed. Since this can be done easily, one seems hardly justified in taking the chance of injury to the eye in this way by not obtaining paralysis of the orbicularis muscle before beginning the operation.

USE OF SEDATIVES

The careful use of sedatives before operation will assist greatly in production of a quiet, tranquil patient. The various sedatives act differently in different persons. In some they increase the excitability and may cause nausea and vomiting, so that it does not seem advisable to use any sedative, particularly in cases of operation on the globe, without one's first ascertaining the action of the drug on that particular patient. Usually the patient is admitted to the hospital at least one day before the operation. On the first night in the hospital a sedative can be given; this allows the operator not only to learn the action of the drug on that patient, but to determine whether the drug acts in the usual manner; besides, it gives the patient a quiet, restful sleep, which is excellent preliminary preparation for the operation. If there

is no undesirable reaction, it is usually safe to use the drug again before the patient goes to the operating room. Many patients, especially the older ones, are accustomed to taking a sedative; if so, it is wise to use the drug to which the patient is accustomed.

Some of the barbituric acid group, such as phenobarbital, sodium amytal and pentobarbital sodium, have a satisfactory action and rarely cause nausea or other undesirable reactions. These drugs have the effect of quieting and calming the patient, which adds greatly to the ease of the operation. Also, they have the added value of counteracting, to some extent, the toxic action of the anesthetic employed.

The dose varies somewhat for different patients. As a test dose, 1½ grains (0.097 Gm.) of phenobarbital may be given the patient before he retires. Then, if satisfactory, a dose of from 1½ to 3 grains (0.097 to 0.195 Gm.) may be given an hour before the operation.

APPROPRIATE METHODS OF ANESTHESIA

In the following paragraphs are listed, in alphabetical order, the different operations on the eye, with a brief indication for each operation of a method for production of complete anesthesia, previously described in detail.

Ablation of Staphyloma.—Sharp pain usually accompanies the sudden decrease of intraocular tension, which often precipitates forcible contraction of the orbicularis muscle, so that complete anesthesia is desirable. An appropriate method of induction of such anesthesia consists in: instillation of anesthetic (page 787), retrobulbar injection within the muscular cone (page 791), induction of akinesia (page 802) and subconjunctival injection, if the eye is inflamed (page 790).

Abscission of Prolapsed Iris.—Instillation anesthesia, induction of akinesia and subconjunctival injection, since the eye is usually inflamed, are indicated. This procedure is usually sufficient, but if the eye is severely inflamed and painful and the wound is not large, injection within the muscular cone should also be made, so that when traction is exerted on the iris no pain will be experienced. The operation can then be completed leisurely and carefully, without danger to the eye.

Blepharoplasty.—The method of anesthesia used will depend on the extent of the involvement and the individual features of the case. By means of infiltration anesthesia and field or nerve block, extensive plastic operations may be performed satisfactorily.

Slitting of the Canaliculus.—The appropriate method consists of instillation of anesthetic (page 787) and infiltration just below the canaliculus.

Canthotomy.—The appropriate method of anesthesia consists of instillation of anesthetic and infiltrations of the subcutaneous and deeper tissues between the external canthus and the orbital margin.

Canthoplasty.—The same method of anesthesia as that used for canthotomy is indicated except that the lids are infiltrated as far as the operation is to extend. If the infiltration is followed by pressure and massage, there will be little distortion of the operative field. Block of the lid (page 801) may be used and thus infiltration of the operative field be avoided, but this is rarely necessary.

Capsulotomy, or Discission of the Capsule.—Instillation anesthesia is usually sufficient for the discission of congenital or juvenile cataracts except with babies and young children, for whom general anesthesia is indicated. For removal of secondary or membranous cataracts, in which the capsule is thick and tough, instillation anesthesia is not sufficient. To avoid the pain caused by traction on the ciliary processes, subconjunctival injections above and below the cornea should be given. Frequently,

patients who have had a discission with instillation anesthesia remark that they did not feel the extraction but that the needling was very painful.

Extraction of Cataract.—Instillation anesthesia, induction of akinesia and retrobulbar injection of the muscular cone are indicated. In cases of dislocated cataract in which the eye is inflamed and painful a subconjunctival injection should also be made to avoid production of pain when the fixation forceps is used.

Cauterization of the Cornea (ulcer; keratoconus).—Instillation anesthesia is usually sufficient except in an operation in which the cornea is to be perforated; then retrobulbar injection of the muscular cone should be made to prevent the sharp pain incident to the sudden evacuation of the anterior chamber. If the eye is inflamed, as with a corneal ulcer, a subconjunctival injection should be given to avoid production of pain when the fixation forceps is used.

Chalazion.—Instillation anesthesia and injection through the conjunctiva of the cul-de-sac into the area corresponding to the chalazion (page 801) constitute the method of anesthesia.

Coloboma and Injuries of the Lids.—Instillation anesthesia and block of the lid (page 801) or infiltration around the area involved are indicated.

Incision for Acute Dacryocystitis.—The procedure is less painful if ethyl chloride is sprayed on a cotton applicator and applied to the site of the incision. On account of the severe induration and swelling, infiltration anesthesia or nerve block is often not advisable.

Dacryocystorhinostomy.—The method of anesthesia consists of block of the infratrochlear (page 789) and the infraorbital (page 799) nerve and use of a nasal pack.

Detachment of the Retina (microdiathermic punctures; ignipuncture).—Instillation anesthesia and injection of the muscular cone (page 791) are indicated.

Ectropion and Entropion.—The method of anesthesia varies according to the operation to be performed. For cauterization, instillation and infiltration anesthesia are sufficient. For the more extensive plastic operations involving the lid, infiltration anesthesia (page 787) or block of the lid (page 801) is necessary.

Enucleation and Evisceration.—Instillation anesthesia, injection of the muscular cone and subconjunctival injection, if the eye is inflamed and painful, are indicated.

Extirpation of the Lacrimal Gland.—Instillation anesthesia and infiltration along the superior temporal margin of the orbit (page 787) are indicated.

Extirpation of the Lacrimal Sac.—Block of the infratrochlear and the infraorbital nerve (page 798) produces complete anesthesia without any disturbance of the anatomic structures around the sac. By the infiltration method the sac can be removed without pain, but if the nasal duct is to be curetted, pain is usually experienced. By use of pressure and massage after infiltration of the tissue over the sac, around the top of the sac and the region about the entrance of the nasolacrimal duct, the anatomic structures and landmarks are disturbed very little.

Foreign Body in the Cornea.—Instillation anesthesia is indicated. One instillation is often sufficient, but the introduction of a few extra drops requires little time and adds greatly to the comfort of the patient and the ease with which the offending particle is removed; in the end it frequently saves time and epithelium.

Intraocular Foreign Body.—The appropriate procedure for induction of anesthesia consists of instillation of anesthetic, induction of akinesia, injection within the muscular cone and subconjunctival injection, if the eye is red and inflamed. When a foreign body is removed through a scleral incision, a retro-

bulbar injection of the anesthetic with an added amount of epinephrine hydrochloride has the advantage of making the eye soft, so that the danger of presentation of the vitreous is lessened.

Glaucoma (Cyclodialysis; Iridectomy; Iridotasis; Lagrange's Operation; Sclerotomy).—Instillation anesthesia, induction of akinesia, injection within the muscular cone and subconjunctival injection are indicated. The last injection should be made if the eye is inflamed and painful, as in cases of acute glaucoma; otherwise the use of the fixaton forceps causes pain.

By the retrobulbar injection the pain incident to a sudden decrease of tension is avoided. Also, a postoperative increase in tension, due to edema of the ciliary processes, or the so-called quiet iritis, is less likely to occur. In some of the aforementioned operations production of akinesia may be superfluous, but if the patient squeezes the eye hard after the incision is made considerable damage may easily be produced; induction of akinesia does no harm and requires only a few minutes.

For the corneoscleral trephination, a 0.5 per cent solution of tetracaine hydrochloride, a 2 per cent solution of butacaine sulfate or an anesthetic that does not dilate the pupil is instilled. Epinephrine hydrochloride is not used until just before the operation is begun.

Subconjunctival injections of a 2 per cent solution of procaine hydrochloride are made at the nasal and the temporal side of the cornea and through the conjunctiva of the upper cul-de-sac deep along the superior rectus muscle.

Injection within the muscular cone gives more complete anesthesia, and if only 1 drop of epinephrine hydrochloride (1:1,000) to 10 cc. of 2 per cent procaine hydrochloride is used, the tension is not lowered enough to prevent prolapse of the iris.

Induction of akinesia is advisable, as the patient's squeezing the eye after the aqueous escapes may injure the lens or cause prolapse of the ciliary processes.

Incision of Hordeolum.—Although this incision is a simple procedure, it is extremely painful. A cotton applicator saturated with ethyl chloride and applied to the site of the incision renders the operation less painful. If there are not too much swelling and induration, often the lid around the hordeolum can be infiltrated so that little pain is experienced; in some cases block of the nerve or lid is most satisfactory (page 801).

Iridectomy; Iridotomy.—Instillation anesthesia, induction of akinesia, injection within the muscular cone and subconjunctival injection, if the eye is inflamed and painful, are indicated. In some iridotomies in which a small incision is made in the cornea, such as that for *iris bombé,* akinesia is not necessary.

Keratoplasty.—Instillation anesthesia, induction of akinesia and injection within the muscular cone are indicated. As cocaine somewhat devitalizes the cornea, its use may account for some of the grafts' becoming cloudy. For this operation use of other anesthetic agents for instillation, such as tetracaine, seems advisable.

Krönlein Operation.—While this operation is usually performed with the patient under general anesthesia, it may also be done with local anesthesia, just as are many intracranial operations.

The type of anesthesia to be used depends to a great extent on the orbital involvement. With large orbital tumors, particularly vascular growths, an orbital block is not advisable. With small tumors, or when the operation is done to relieve exophthalmos, the procedure may be accomplished with a field block surrounding

the area of operation. After the bone flap has been raised, intraorbital injections can be used as necessary, the indication depending on the amount of orbital interference.

Operations on Muscles (Advancement; "Cinching" or Tucking Operations; Recession; Resection).—Instillation of anesthetic and injection within the muscular cone constitute the method used. The paresis of the muscles caused by the retrobulbar injection is an objection but is not so serious as with general anesthesia. However, before the operation the surgeon has usually estimated the amount of correction necessary and has decided on just how much advancement, resection or recession of the muscle he will produce, so that the paresis is not a serious handicap. A deep injection may be made along the muscle, but it does not prevent the pain caused by traction on other muscles, and frequently the injected solution balloons the surrounding tissues, so that the operation is made more difficult. Also, an injection within the cone is as easy and safe as a deep injection along the muscle.

Tenotomy of the rectus muscles can be performed satisfactorily with instillation anesthesia, but usually it causes some pain, which can be avoided by either injection within the muscular cone or injection in the vicinity of the muscle to be tenotomized; if massage with pressure is employed, the anatomic relations are not disturbed enough to interfere with a satisfactory tenotomy.

Tenotomy of the Inferior Oblique Muscle.—1. A wheal is raised in the vicinity of the inferior nasal margin of the orbit (page 789). 2. Tissues beneath the skin and the deeper tissues along the orbital margin in the vicinity of the attachment of the inferior oblique muscle are infiltrated (page 787). If the tenotomy is to be done through the conjunctiva, instillation anesthesia should be used, and the injection along the orbital margin may be made through the conjunctiva of the lower cul-de-sac.

Opticociliary Neurotomy.—The method employed is instillation of anesthetic, injection within the cone and subconjunctival injection, since the eye is usually inflamed and painful.

Paracentesis of the Cornea and Sclera.—The eye is usually inflamed, and the operation, although simple, is generally extremely painful unless complete anesthesia is obtained. The method consists in instillation of anesthetic, injection within the muscular cone, subconjunctival injection, if the eye is inflamed (otherwise the use of fixation forceps is painful), and, in some cases, induction of akinesia.

Plastic Operations.—Since plastic operations vary to such a degree with each patient, it is not practical to attempt to outline a definite procedure for induction of anesthesia. While some extensive plastic operations require general anesthesia, many can be performed with local anesthesia by means of infiltration, nerve block and field block (page 789). Scar tissue cannot be satisfactorily infiltrated, but unless it is extensive, the area can usually be rendered anesthetic by the use of field or nerve block.

Probing of the Lacrimal Duct.—Instillation anesthesia and the injection of several drops of the anesthetic solution into the lacrimal sac somewhat relieve the pain, but block of the infratrochlear and the anterior superior alveolar nerve (page 796) renders the procedure practically painless. For passage of small probes, in which little pressure is exerted, instillation into the conjunctival and lacrimal sacs is usually sufficient, but insertion of a large probe, if anesthesia is not adequate, is usually very painful.

Pterygium.—Instillation anesthesia and subconjunctival injection under the pterygium, and below the cornea if transplantation is to be made below, are indicated.

Ptosis.—The operations for ptosis are numerous, but the majority can be performed with the aid of local anesthesia, particularly if the patient is not too young.

Here, again, the procedure for induction of anesthesia depends on the operation to be performed. For operations that involve the superior rectus muscle, such as the Motais operation, the following procedure is adequate: instillation of anesthetic, injection within the muscular cone, subconjunctival injection into the upper cul-de-sac and infiltration under the skin of the lid, where the sutures pass through the skin.

Saemischi's Keratotomy.—Instillation anesthesia, injection within the cone and subconjunctival injection, to avoid pain on use of the fixation forceps, are adequate.

Tattooing.—Instillation anesthesia is adequate.

Trachoma.—Expression: Instillation anesthesia and subconjunctival injection into the cul-de-sac of a large amount of anesthetic solution are indicated. In young children and in some adults general anesthesia is usually necessary, as the operation is extremely painful.

Combined Excision of Fornix and Tarsus: Instillation anesthesia and subconjunctival injection of a large amount of anesthetic solution into the cul-de-sac are adequate.

CONCLUSION

Practically all ophthalmic operations may be painlessly and safely performed with use of local anesthesia provided the anesthetic is carefully and properly administered.

Correspondence

EMMETROPIA

To the Editor:—The concept of emmetropia, though it is fundamental in the ophthalmologist's work, is hardly as simple and fixed as textbook definitions seem to imply. As evidence of this one may refer to an article by Dr. Edwin S. Munson (Emmetropia, ARCH. OPHTH. **29:** 109 [Jan.] 1943). Several points relative to emmetropia not touched on in that article are, I believe, of sufficient interest to be mentioned.

The aberrations of the eye play an important role not only in determination of visual acuity but in the very definition of emmetropia. Gullstrand, for example, made his emmetropic eye 1.00 D. hyperopic for paraxial rays. To understand fully the implication of this, one must visualize how the light from an object point is acted on by the eye as the light passes through the different mediums. In essence, every pencil of light from an object which passes through the pupil reaches the vitreous as a more or less irregular, cone-shaped bundle of rays—call it conoid for short—the impact of which on the retina produces a diffusion spot. The rays which compose the outer layers of the conoid form innumerable and variously placed focal points with one another before and after they strike the axis. The aggregate of these multiple and scattered focal points form the so-called caustic. In the usual positive spherical aberration, the rays immediately around the axis, the so-called paraxial rays, focus farther back on the axis than the marginal rays, those passing through the edge of the pupil.

The cross section of the conoid where it strikes the retina forms a diffusion spot, in which the concentration of light will never be uniform throughout. The spot differs in size, shape and brightness distribution according to the section of the conoid intercepted by the retina. Somewhere between the axial focus of the marginal rays and the axial focus of the paraxial rays is the "circle of least confusion." This forms the narrowest part, or the "waist," of the conoid. If the retina intercepts the conoid at or near the marginal focus (therefore in front of the circle of least confusion), the diffusion patch on the retina will show the greatest concentration of light near the periphery of the patch. If the retina intercepts the conoid at or near the paraxial focus, the diffusion patch on the retina will show the greatest concentration of light near the center of the patch. If the retina intercepts the conoid at the circle of least confusion, the difference between the light and the dark areas in the patch is generally least and the patch is more nearly uniformly bright.

It seems that the cross section of the conoid which gives best vision, and to which the eye tends to adapt itself, varies not only in different persons but in the same person, the section depending on the nature of the object viewed. Gullstrand stated that the eye adapts itself to a section of the conoid about 1 D. in front of the paraxial focus. Therefore his "emmetropic" eye, emmetropic in the sense that it is best adapted for clear distant vision on the usual charts, has 1 D. of hyperopia for paraxial rays.

On the other hand, examination by the ophthalmoeikonometer shows that an eye which has been made emmetropic in the sense that it is best adapted for distant vision on the Snellen chart is about 0.50 D. myopic for a target consisting of a minute point of light. This can be explained on the supposition that when made "emmetropic" for the Snellen chart the eye chooses a section of the conoid at or near the paraxial focus, where the greatest concentration of light is near the center, but when focusing on a bright point of light it chooses a section of the conoid corresponding to the "waist," or circle of least confusion. This point is about 0.50 D. in front of the paraxial focus. Luckiesh and Moss, in making tests with an instru-

ment of their own invention, the sensitometer, stated: "In the typical emmetropic subject, during a state of complete rest, the eyes are about 0.75 D. myopic." This bears out the findings of the Dartmouth school. This discrepancy they explained on the basis of negative relative accommodation; more likely it is due to the eye's choosing a different section of the conoid for best vision of different targets.

Thus the concept of emmetropia based on what actually takes place in the act of seeing involves consideration of the methods of testing, the size of the pupil ("An eye that was emmetropic with a pupil of medium size would have to show hypermetropia amounting to 1 diopter with an infinitely small pupil" [Gullstrand]), the nature of the test object, the relative illumination of the test object and the background and possibly other factors. One of the best articles on the effect of the retinal diffusion patches on vision and visual acuity is a paper by Dr. Walter B. Lancaster (Stigmatoscopy, *Tr. Am. Ophth. Soc.* **32:** 130, 1934). The discussion and illustrations, presented in Dr. Lancaster's lucid, inimitable way, will be an eye opener to persons whose concept of emmetropia is based on the old, familiar diagram of parallel rays meeting at the retina.

JOSEPH I. PASCAL, M.D., New York.

37 West Ninety-Seventh Street.

CONVERSION FORMULA FOR SNELLEN SYMBOLS

To the Editor.—Dr. Gabriel's equations, which appeared in the July issue (ARCH. OPHTH. **30:** 138, 1943) are simple and correct for conversion from the Snellen to the Navy terminology. Why not make the method still simpler?

$$6/7.5 = 20/25 = 4/5 = ?/20 = 16/20$$
$$20/30 = 2/3 = ?/20 = 13/20$$

This soon forms a picture in the examiner's brain, and the process becomes semiautomatic.

D. G. ALLEN, M.D., Cleveland.

1021 Prospect Avenue

News and Notes

GENERAL NEWS

American Board of Ophthalmology.—An examination will be held in New York on June 3 and 4, 1944 and in Chicago on Oct. 5, 6 and 7, 1944.

Further information with respect to examinations may be obtained from the secretary-treasurer, Dr. S. Judd Beach, P. O. Box 1940, Portland 2, Maine.

The other officers for 1944 are: chairman, John Green, M.D., St. Louis; vice chairman, Frederick C. Cordes, M.D., San Francisco, and assistant secretary, Theodore L. Terry, M.D., Boston.

Ophthalmic Gold Medal.—The Ophthalmological Society of Egypt offers a yearly prize, consisting of a gold medal of the value of £20 (English), for the most valuable contribution presented before the annual congress of the society. This competition is open to members of the society, and the manuscript is to be sent in by December.

SPECIAL NEWS

Course for Orthoptic Technicians.—The Rochester (N. Y.) Orthoptic Center (approved by the Monroe County Medical Society) offers a course for orthoptic technicians. Young women desirous of making orthoptics a career and of preparing for the examinations of the American Orthoptic Council may get full particulars from Mrs. Margaret Lundean, orthoptic technician, 31 North Goodman Street, Rochester, N. Y.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

A QUANTITATIVE STUDY OF THE CELLS AND FIBERS IN THE NUCLEUS: NERVE COMPLEXES OF THE FOURTH AND SIXTH CRANIAL NERVES. R. D. HARLEY, Am. J. Ophth. **25:** 1029 (Sept.) 1942.

Harley collected specimens of the nerves and nuclei from 32 normal human brains. From a study of these specimens, he concludes:

"Since there are clearly more cells in the so-called nuclei than motor fibers in the peripheral nerves, it is logical to assume that the motor nuclei of the fourth and sixth cranial nerves send fibers not only peripherally into these nerves, but centrally to other regions, such as to other cranial nuclei, probably via the median longitudinal fasciculus. Some of the association mechanism (association centers) may thus be within the confines of the so-called nuclei of the ocular nerves."

W. S. REESE.

THE AQUEOUS VEINS. K. W. ASCHER, Am. J. Ophth. **25:** 1174 (Oct.) 1942.

In his summary, Ascher states that aqueous veins are biomicroscopically visible pathways with appearance of blood vessels, containing a clear, colorless fluid or diluted blood and intercalated, probably via Schlemm's canal, between intraocular fluid, on the one side, and conjunctival and subconjunctival veins, on the other. Anatomically they are connected with, or are a part of, the intrascleral network. He describes tests that prove this and expresses the opinion that these aqueous veins confirm Leber's theory of a continuous elimination of fluid from the intact human eye.

W. S. REESE.

Aqueous Humor

THE INTRASCLERAL VASCULAR PLEXUS AND ITS RELATIONS TO THE AQUEOUS OUTFLOW. M. U. TRONCOSO, Am. J. Ophth. **25:** 1153 (Oct.) 1942.

Troncoso compares the anatomy of the anterior chamber of different species of animals. He concludes that in primates there is not a blood-aqueous barrier and that aqueous passes directly through the collectors into the blood stream.

W. S. REESE.

SULFONAMIDE CONTENT OF AQUEOUS HUMOR FOLLOWING CONJUNCTIVAL APPLICATION OF DRUG POWDERS. E. GALLARDO and R. THOMPSON, Am. J. Ophth. **25:** 1210 (Oct.) 1942.

Gallardo and Thompson give the following summary:

"Acute inflammation produced by mustard oil [allyl isothiocyanate] in the conjunctiva or by streptococcal infection of the anterior chamber increased the passage of sulfanilamide into the aqueous fluid. The increase may have been due entirely to injury of the corneal epithelium, since intraocular inflammation by the intravenous injection of killed typhoid bacilli definitely diminished passage of the drug. Even with this inflammation, however, effective aqueous fluid concentrations could often be demonstrated.

"Of the drugs tested by local application, the higher levels in the aqueous humor were obtained with sulfanilamide."

W. S. REESE.

Conjunctiva

ACUTE FOLLICULAR CONJUNCTIVITIS RESEMBLING BÉAL'S TYPE. M. P. KOKE, Am. J. Ophth. **25:** 1100 (Sept.) 1942.

Koke describes an epidemic of acute follicular conjunctivitis among 66 aircraft workers. The cardinal characteristics of the disease were (1) acute or subacute unilateral onset with profuse lacrimation; (2) absence of exudate; (3) severe hyperemia of the conjunctiva; (4) folliculosis, which was most marked in the conjunctiva of the inferior fornix; (5) slightly tender preauricular adenitis; (6) swelling of the lids; (7) complete resolution in seven to twenty-one days; (8) absence of inclusion bodies or pathogenic bacteria in scrapings from the conjunctiva, and (9) abundance of lymphocytes in the conjunctival scrapings. Seven patients showed superficial keratitis. Koke suggests that this condition is due to a virus.

W. S. REESE.

Congenital Anomalies

A RARE CONGENITAL OCULAR ABNORMALITY. G. S. PENDSE, Indian J. Ophth. **4:** 1 (Jan.) 1943.

A girl, born at full term, would not open her eyes at birth, and examination revealed absence of the eyeballs though a bluish swelling within the right orbit showed that the condition was not anophthalmos. Examination a few weeks later showed that the swelling was increasing under the skin, which was bluish, and revealed another swelling in the left lower lid. Transillumination revealed that both were transparent. There were no other ocular or extraocular abnormalities. The diagnosis of bilateral microphthalmos with cysts, though the most probable one, cannot be verified without pathologic examination of the cysts. The article is illustrated.

W. ZENTMAYER.

Cornea and Sclera

CORNEAL PERMEABILITY. K. C. SWAN and N. G. WHITE, Am. J. Ophth. **25:** 1043 (Sept.) 1942.

Swan and White describe their experiments on corneal permeability and state that permeability is not a simple matter of diffusion but a complex process affected by multiple interrelated factors and that the concept that the epithelium acts as a barrier limiting the penetration of all substances into the cornea is erroneous. They conclude:

"In evaluation of the ocular effects of drugs administered by instillation, penetration rate under varying conditions as well as pharmacologic potency must be considered. The difference in effectiveness of topical anesthetics may be due principally to differences in penetration rate into the cornea."

W. S. REESE.

EPIDEMIC KERATOCONJUNCTIVITIS. M. J. HOGAN and J. W. CRAWFORD, Am. J. Ophth. **25:** 1059 (Sept.) 1942.

Hogan and Crawford give the following summary and conclusions:

"This paper reports the occurrence of a severe epidemic of keratoconjunctivitis occurring in, but not limited to, shipyard workers. In all 125 cases are reported.

"The disease we have described is a superficial keratoconjunctivitis which, because of its epidemic form, should be called epidemic keratoconjunctivitis. This disease has been described under various names such as keratitis nummularis (Dimmer) and some forms of keratitis disciformis associated with superficial macular lesions. From its appearance and history the disease suggests a relationship to Béal's form of conjunctivitis. Superficial epithelial erosions should not be classed with the disease under discussion.

"There is great variation in the clinical picture, even during epidemics. Seventy-five per cent of the conjunctival infections are unilateral. Diagnostic in the early states are the glassy edema of the conjunctiva, involvement of the regional lymph apparatus, the lymphocytic type of conjunctival exudate, tiny petechial hemorrhages, pseudomembrane formation, and the lack of discharge. The severe conjunctivitis may or may not be followed by keratitis. Keratitis occurs in about 75 per cent of the cases, though this figure varies in different epidemics. At times keratitis occurs without a history of preceding conjunctivitis.

"The disease is most likely caused by a virus, the nature of which is unknown but which may be related to the herpes-facialis group. The disease is not highly infectious. It is transmitted by direct contact to susceptible persons, and individual susceptibility varies.

"Therapy is of little avail except possibly in the early stages and in milder cases. The disease is self-limited.

"The ultimate prognosis is good as regards vision."

W. S. REESE.

Experimental Pathology

LYMPHOGRANULOMA-VENEREUM LESIONS OF THE EYES. C. ESPÍLDORA and W. E. COUTTS, Am. J. Ophth. **25:** 916 (Aug.) 1942.

The authors review the literature concerned with experimental lesions in animals and lesions in man and make the following conclusions:

"(1) The eye may be the site of lymphogranuloma-venereum lesions during any one of the periods of this disease.

"(2) Any structure of the eye may be involved by the process.

"(3) All structures in direct contact with the exterior may serve as the site of inoculation for the virus of lymphogranuloma venereum."

W. S. REESE.

General Diseases

THE RELATION OF RIBOFLAVIN TO THE EYE. A. PIRIE, Brit. J. Ophth. **27:** 291 (July) 1943.

The subject is reviewed under the following heads: properties of riboflavin; occurrence of riboflavin in the body; flavoprotein enzymes, and relation of riboflavin to the eye, general effects of riboflavin deficiency in animals, and effects of deprivation of riboflavin on the eyes of experimental animals.

Pirie considers it extremely interesting that the ocular signs of riboflavin deficiency are among the earliest to develop and may be used in conjunction with changes in the skin and tongue in making an early diagnosis of the deficiency.

W. ZENTMAYER.

Glaucoma

ROENTGEN THERAPY OF GLAUCOMA. M. F. TERRIZZANO and A. M. J. TERRIZZANO, Semana méd. **49:** 1354 (Dec. 3) 1942.

The Terrizzanos employed roentgen irradiation in 10 cases of glaucoma. In 1 case the disease was of traumatic origin; in 6 it was painful and chronic; in 2 hemorrhages occurred, and in 1 the glaucoma was accompanied by hypertensive iritis. Improvement resulted in all cases. Pain and lacrimation either disappeared or improved. Keratoconjunctival inflammation disappeared rapidly, and no disagreeable reaction was observed. In only 1 of the 10 cases did a relapse occur, after seven months, and two new irradiations were effective in counteracting the condition. The authors suggest that the effect of roentgen therapy is due to complex action on intraocular osmotic phenomena, modifications in the capillary circulation, tissue ionization, variations in the acid-base and colloid equilibrium

and direct action on the neural terminations. Irradiation using a tension of 140 kilovolts, with 10 milliamperes, at a distance of 50 cm. and filtration through 0.25 mm. of copper and 1 mm. of aluminum gives the best results.

J. A. M. A. (W. ZENTMAYER)

Injuries

TETANUS FOLLOWING EYE INJURY. J. O. WETZEL, Am. J. Ophth. **25**: 933 (Aug.) 1942.

Wetzel reports the case of a farmer who sustained a perforating laceration of the eyeball, with prolapsed iris, as a result of an injury from a nail while he was working in his barn. Tetanus developed, but he recovered, the eye having been enucleated. Wetzel reviews 30 case reports in the literature and urges the routine use of tetanus antitoxin because of the high mortality.

W. S. REESE.

PIECE OF GLASS MOVABLE IN THE EYEBALL FOR TWENTY-THREE YEARS: REPORT OF A CASE. R. CAUER, Klin. Monatsbl. f. Augenh. **106**: 91 (Jan.) 1941.

The rimless noseglass of a youth aged 15 was struck by a toy arrow and a splinter entered his eye. A penetrating wound was observed in the sclera 9 mm. nasal to the limbus. Apparently complete recovery with perfect vision was obtained two months later. No trace of the splinter could be observed after the vitreous had cleared. The patient returned after twenty-two years, during which period his eye had been free from any disturbance. Vision in that eye had become blurred, and congestion was noted on the morning of his visit. A few days later the splinter, 4 mm. long, presented at the floor of the anterior chamber. It had slowly traveled a distance of 9 mm. The eye recovered promptly after extraction of the splinter.

K. L. STOLL.

LOCALIZATION OF FOREIGN BODIES IN POSTERIOR WALL OF THE EYEBALL, AND THEIR OPERATIVE REMOVAL BY USE OF THE SCLERAL LAMP: REPORT OF A CASE. H. COLDITZ, Klin. Monatsbl. f. Augenh. **106**: 94 (Jan.) 1941.

The increasing frequency of nonmagnetic foreign bodies in the eye requires new methods for their removal. A number of these technics are discussed and evaluated. Colditz describes his own method, by means of which he extracted a piece of copper, a portion of the cartridge, from the eye of a patient. It had been in the eye about five weeks and had produced exudation in the periphery of the fundus, decrease of vision and contraction of the field of vision. The new method, consisting of inflation of air into Tenon's capsule and simultaneous use of Comberg's prothesis, allows exact intrabulbar and extrabulbar localization of splinters. Diascleral illumination proved of great value. The splinter was removed, as described in detail, and perfect recovery was obtained, with four-fifths normal vision and a normal field of vision.

K. L. STOLL.

Lens

NOTE ON INTRACAPSULAR EXTRACTIONS OF CATARACT BY WEBER'S LOOP AS A ROUTINE METHOD. D. D. SATHAYE, Indian J. Ophth. **4**: 9 (Jan.) 1943.

The usual corneal incision is made with a long, narrow conjunctival bridge above, and iridectomy is performed. A Weber loop is introduced and directed obliquely backward through the incision until it reaches a point in the middle of the vitreous and about opposite the posterior pole of the lens. By depression of its handle, it is then carried forward, so that the lens is pressed against the posterior surface of the cornea, between the cornea and the loop. In other words, the

lens is lifted out of the eye by the loop. This method has been used in a series of 38 consecutive cases, with good results. In 1 case there was an excessive escape of vitreous, and in another case the capsule of the lens was ruptured.

The article is well illustrated.

W. ZENTMAYER.

Ocular Muscles

RECESSION OF THE TROCHLEA IN OVERACTION OF THE SUPERIOR OBLIQUE. W. L. HUGHES and D. W. BOGART, Am. J. Ophth. **25:** 911 (Aug.) 1942.

Hughes and Bogart briefly discuss overaction of the superior oblique muscle and suggest a simple effective operation of recession of the trochlear as a remedy. Two cases are reported.

W. S. REESE.

EXPERIMENTAL TRANSPOSITION OF EXTRAOCULAR MUSCLES IN MONKEYS. P. J. LEINFELDER and N. M. BLACK JR., Am. J. Ophth. **25:** 974 (Aug.) 1942.

This is the authors' second article on this subject, and they conclude that the superior oblique muscle alone does not appear to have any special coordinating function that cannot be ascribed to any other muscle. They suggest there may be a relation between the amount of involvement of Tenon's capsule in surgical intervention and recovery of coordination.

W. S. REESE.

Orbit, Eyeball and Accessory Sinuses

THROMBOPHLEBITIS OF THE CAVERNOUS SINUS: RECOVERY. A. R. C. DOORLY, Brit. M. J. **1:** 42 (Jan. 9) 1943.

A healthy married woman aged 56 had a nasal furuncle inside the left nostril. There was the usual edematous swelling of the adjacent cheek, with intense pain and slight fever. She was treated with sulfathiazole, one tablet (0.5 Gm.) every four hours, and glycerin packs were advised for the interior of the nose. On the following day there was more swelling, the temperature was 101 F. and pus was being discharged from the furuncle. Two days later the left eye was proptosed, both lids were greatly swollen and the right eye was commencing to be involved. The patient complained of seeing double on looking to the left. There was some rigidity of the neck muscles, and the temperature rose to 103 F. A blood culture showed Staphylococcus aureus of the same type as that found in the nasal lesion. She was treated with transfusion of 400 cc. of whole blood and intramuscular injection of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) alternating with oral administration of the drug, 4 Gm. being given by each route within the first twenty-four hours. After two days the temperature began to fall and the general condition improved. The chemotherapy was continued, though after three days the dose was reduced. The next day there was a sharp rise of temperature, and a second transfusion of blood was given. This was followed by improvement, but there was a third relapse. Another blood transfusion was given and the sulfathiazole treatment continued. During the next four days the patient remained very ill, with an extensive rash and severe headache, but the local condition of the eyes showed improvement. As the rash was so distressing and the patient by then had taken 95 Gm. of sulfathiazole, the chemotherapy was discontinued. After this the temperature gradually came down, the rash faded and the swelling left the lids. Headache gradually ceased, and convalescence began.

It has been generally recognized that free incision is dangerous and apt to lead to rapid dissemination of the infection. Hence the method of treatment reported, offering a good chance of recovery, is extremely gratifying. It is difficult to say whether the blood transfusions or the sulfathiazole played a greater part in the cure of the condition, but the author thinks the combination important. The case proved conclusively the great value of sulfathiazole in the treatment of

serious staphylococcic infections and showed that the toxicity of this drug is only half that of sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine).

ARNOLD KNAPP.

Tumors

FIVE HUNDRED MELANOMAS OF THE CHOROID AND CILIARY BODY FOLLOWED FIVE YEARS OR LONGER. G. R. CALLENDER, H. C. WILDER and J. E. ASH, Am. J. Ophth. **25:** 962 (Aug.) 1942.

The authors give the following summary of their article:

"There are 1,600 cases of malignant intraocular melanoma on record in the American Registry of Ophthalmic Pathology. Only eight of these are in Negroes. There is a slight preponderance of males over females. The tumor is one peculiar to adult life and appears but rarely in the earlier decades. Five hundred patients suffering from melanoma of the choroid and ciliary body have been followed five years or longer, or have died as a result of metastases before the expiration of that time. The tumors in this group have been classified by cell type and fiber content, and the following corollaries have been drawn: The pure spindle-cell types, particularly subtype A, are of a relatively low degree of malignancy, and an increased degree of malignancy is associated with the presence of epithelioid cells; a low degree of malignancy is usually associated with a heavy fiber content.

"Although there appears to be an increased lethality with increased pigment content, it is felt that there is not enough variation in death percentages to justify conclusions until a larger number of followed cases has been accumulated. The total number of metastatic deaths is 239 among the cases followed five years or longer, or 48 per cent. This percentage increased to 66 in 200 of these cases followed 10 years or longer."

W. S. REESE.

Vision

COMPARISON OF CASES OF ATYPICAL AND TYPICAL ACHROMATOPSIA. L. L. SLOAN and S. M. NEWHALL, Am. J. Ophth. **25:** 945 (Aug.) 1942.

Sloan and Newhall report their studies on 3 cases of achromatopsia. They discuss the different varieties of this condition and comment on the possible causes and the theories which have been advanced to explain them.

W. S. REESE.

MEASURE OF NIGHT VISION. N. B. HARMAN, Brit. M. J. **1:** 43 (Jan. 9) 1943.

There are several tests for night vision. Some are distinctly ingenious, but with some there is no definite measure of the minimum light necessary for vision in the particular subject tested, which the author regards as the most important point. This measure should be given by a direct reference to the scientific standard of candle power, and it should be ascertained and recorded in terms which can be used by any ophthalmic surgeon who repeats the process.

The earliest tests of night vision measured the time required by the subject to become adapted to the dark, but adaptation tests do not give a definite measure of the minimum light required to see an object or objects. The objection to Dr. W. D. Wright's and Major Rycroft's tests is that they do not give a definite measure of the lighting, so that the results are not exactly comparable.

The disk-spotting test (Harman, N. B.: Testing Night Vision, *Brit. M. J.* **2:** 347 [Sept. 6] 1941) is planned on a direct measurement of light. The light is reflected from illuminated disks on a black velvet background, and the measure is gained by finding the distance at which the disks can be counted. This distance is designated in fractions of 5, and the fractional figure indicates the condition of the patient's night vision, which is fully explained in this article. The illumination is from the standard candle, so that there is exactness of the light used and of its measurement and the returns of the examination are definite.

ARNOLD KNAPP.

Book Reviews

A Hundred Years of Medicine. By C. D. Haagensen and Wyndham E. B. Lloyd. Price, $3.75. Pp. 444, with 42 illustrations. New York: Sheridan House, Inc., 1943.

This book was first published in an English edition by Dr. Wyndham E. B. Lloyd, a specialist in public health in England, and now appears in an American edition. The important medical advances are traced through the years, and to simplify the presentation the authors consider each contribution to medicine separately. The American edition differs much from the preceding one; while part I, which deals with the historical background, and chapters 10 to 19 of part II, on infectious diseases, by Dr. Lloyd, are unchanged except for some additions, the rest of this new edition has been rewritten by the American editor, Dr. C. D. Haagensen, surgeon and pathologist to Columbia University College of Physicians and Surgeons.

"A Hundred Years of Medicine" is written especially for the layman and the beginner in medicine. The authors have admirably succeeded in presenting a book "on the face of diseases," relieved of the dry facts of anatomy, physiology and pathology, which should appeal to the beginner and can be read with interest by the practicing physician, especially by one, like the specialist, whose work has become confined to narrower lines. The book is divided as follows: part I, medicine up to a hundred years ago; part II, medical science during the last hundred years; part III, surgery during the last hundred years, and part IV, the new social aspect of medicine, with a bibliography and an index in conclusion.

It is difficult to pick out outstanding chapters, as all are excellent. The facts are presented in an unusually interesting manner so that the reading becomes fascinating and absorbing. The subjects are completely and successfully presented, and the personalities who took an active part in the remarkable development of medicine are admirably described. The book ends with a view to the future, and the difficulties and advantages of group practice are discussed. This book is a fascinating contribution to medical history and can be highly recommended.

ARNOLD KNAPP.

CORRECTION

In the article by Dr. Otto Lowenstein and Dr. Isadore Givner entitled "Pupillary Reflex to Darkness," in the November issue (ARCH. OPHTH. **30:** 603, 1943), the term "rotary nystagmus" in line 20 on page 608 should read "retraction nystagmus.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 17 Horseferry Rd., London, England.

Pan-American Congress of Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretaries: Dr. Conrad Berens, 35 E. 70th St., New York. Dr. M. E. Alvaro, 1511 Rua Consolacão, São Paulo, Brazil.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. I. Iwre, Budapest.
Assistant Secretary: Dr. Stephen de Grósz, University Eye Hospital, Máriautca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

*Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. A. MacRae, 6 Jesmond Rd., Newcastle-upon-Tyne, England.
Secretary: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation.
Time: October to April.

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the secretary, Dr. Mohammed Khalil.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Mr. P. G. Doyne, 60 Queen Anne St., London, W. 1, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford, England. Time: July 8-9, 1943.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Col. F. A. Juler, 96 Harley St., London, W. 1, England.
Secretary: Dr. Harold Ridley, 60 Queen Anne St., London, W. 1, England.

SÃO PAULO SOCIETY OF OPHTHALMOLOGY

President: Dr. W. Belfort Mattos, Caixa Postal, 4086, São Paulo, Brazil.
Secretary: Dr. Silvio de Almeida Toledo, Enfermaria Santa Luzia, Santa Casa de Misericordia, Cesario Motta, St. 112, São Paulo, Brazil.

SOCIEDAD ARGENTINA DE OFTALMOLOGIA

Chairman: Dr. Jorge Malbrán, Buenos Aires.
Secretary: Dr. Benito Just Tiscornia, Santa Fe 1171, Buenos Aires.

SOCIEDAD OFTALMOLOGIA DEL LITORAL, ROSARIO (ARGENTINA)

President: Prof. Dr. Carlos Weskamp, Laprida 1159, Rosario.
Secretary: Dr. Juan M. Vila Ortiz, Córdoba 1433, Rosario.
Place: Rosario. Time: Last Saturday of every month, April to November, inclusive.
All correspondence should be addressed to the President.

SOCIEDADE DE OPHTHALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

Società Oftalmologica Italiana

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 Ill tr., Stockholm, Sö.

Tel Aviv Ophthalmological Society

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Conrad Berens, 35 E. 70th St., New York City.
Secretary: Dr. R. J. Masters, 23 E. Ohio St., Indianapolis.
Place: Chicago. Time: June 12-16, 1944.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
President-Elect: Dr. Lawrence T. Post, Metropolitan Bldg., St. Louis.
Executive Secretary-Treasurer: Dr. William L. Benedict, 101–1st Ave. Bldg., Rochester, Minn.

American Ophthalmological Society

President: Dr. John Green, 3720 Washington Ave., St. Louis.
Secretary-Treasurer: Dr. Walter S. Atkinson, 129 Clinton St., Watertown, N. Y.
Place: Hot Springs, Va. Time: May 29-31, 1944.

Association for Research in Ophthalmology, Inc.

Chairman: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. Brittain F. Payne, School of Aviation Medicine, Randolph Field, Texas.

Canadian Medical Association, Section on Ophthalmology

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

Canadian Ophthalmological Society

President: Dr. Colin A. Campbell, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. Kenneth B. Johnston, Suite 1, 1509 Sherbrooke St. W., Montreal.

National Society for the Prevention of Blindness

President: Mr. Mason H. Bigelow, 1790 Broadway, New York.
Secretary: Miss Regina E. Schneider, 1790 Broadway, New York.
Executive Director: Mrs. Eleanor Brown Merrill, 1790 Broadway, New York.

SECTIONAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. N. Zwaifler, 46 Wilbur Ave., Newark.
Secretary: Dr. William F. Keim Jr., 25 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Paul A. Chandler, 5 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Merrill J. King, 264 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St. Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. D. H. O'Rourke, 1612 Tremont Pl., Denver.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. L. McCoy, 1317 Marion St., Seattle, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 301 Stimson Bldg., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month except June July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. Sheldon Clark, 27 E. Stephenson St., Freeport, Ill.
Secretary-Treasurer: Dr. Harry R. Warner, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. M. H. Pike, Midland, Mich.
Secretary-Treasurer: Dr. R. H. Criswell, 407 Phoenix Bldg., Bay City, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. C. Decker, 515 Francis Bldg., Sioux City, Iowa.
Secretary-Treasurer: Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. John H. Burleson, 414 Navarro St., San Antonio, Texas.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville, S. C.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. H. L. Brehmer, 221 W. Central Ave., Albuquerque, N. Mex.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. W. M. Dodge, 716 First National Bank Bldg., Battle Creek.
Secretary-Treasurer: Dr. Kenneth Lowe, 25 W. Michigan Ave., Battle Creek.
Time: Last Thursday of September, October, November, March, April and May

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Ray Parker, 218 Franklin St., Johnston, Pa.
Secretary-Treasurer: Dr. J. McClure Tyson, Deposit Nat'l Bank Bldg., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Raymond C. Cook, 701 Main St., Little Rock.
Secretary: Dr. K. W. Cosgrove, Urquhart Bldg., Little Rock.

Colorado Ophthalmological Society

President: Dr. C. A. Ringle, 912–9th Ave., Greeley.
Secretary: Dr. W. A. Ohmart, 1102 Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. F. L. Phillips, 405 Temple St., New Haven.
Secretary-Treasurer: Dr. W. H. Turnley, 1 Atlantic St., Stamford, Conn.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. E. N. Maner, 247 Bull St., Savannah.
Secretary-Treasurer: Dr. C. K. McLaughlin, 567 Walnut St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. F. McK. Ruby, Union City.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. K. Von Lackum, 117–3d St. S. E., Cedar Rapids.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Val H. Fuchs, 200 Carondelet St., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Robert H. Fraser, 25 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. G. Laird, 114 Fulton St., Grand Rapids.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Secretary: Dr. William A. Kennedy, 372 St. Peter St., St. Paul.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. William Morrison, 208 N. Broadway, Billings, Mont.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

Nebraska Academy of Ophthalmology and Otolaryngology

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N St., Lincoln.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. B. E. Failing, 31 Lincoln Park, Newark.
Secretary: Dr. George Meyer, 410 Haddon Ave., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Searle B. Marlow, 109 S. Warren St., Syracuse.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. Hugh C. Wolfe, 102 N. Elm St., Greensboro.
Secretary: Dr. Vanderbilt F. Couch, 105 W. 4th St., Winston-Salem.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. T. W. Buckingham, 405 Broadway, Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 516–6th St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Paul Neely, 1020 S. W. Taylor St., Portland.
Secretary-Treasurer: Dr. Lewis Jordon, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. L. Sanders, 222 N. Main St., Greenville.
Secretary: Dr. J. H. Stokes, 125 W. Cheves St., Florence.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. F. H. Rosebrough, 603 Navarro St., San Antonio.
Secretary: Dr. M. K. McCullough, 1717 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. Everett B. Muir, Boston Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Earl H. Phillips, 623 Judge Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Mortimer H. Williams, 30½ Franklin Rd. S. W., Roanoke.
Secretary-Treasurer: Dr. Meade Edmunds, 34 Franklin St., Petersburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. L. Mather, 39 S. Main St., Akron, Ohio.
Secretary-Treasurer: Dr. V. C. Malloy, 2d National Bank Bldg., Akron, Ohio.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. B. M. Cline, 153 Peachtree St. N. E., Atlanta, Ga.
Acting Secretary: Dr. A. V. Hallum, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., fourth Monday of each month, from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. Luther E. Wilson, 919 Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. William B. Agan, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Benjamin C. Rosenthal, 140 New York Ave., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Walter F. King, 519 Delaware Ave., Buffalo.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Vernon M. Leech, 55 E. Washington St., Chicago.
Secretary: Dr. W. A. Mann, 30 N. Michigan Ave., Chicago.
Place: Chicago Towers Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati General Hospital Ophthalmology Staff

Chairman: Dr. D. T. Vail, 441 Vine St., Cincinnati.
Secretary: Dr. A. A. Levin, 441 Vine St., Cincinnati.
Place: Cincinnati General Hospital. Time: 7:45 p. m., third Friday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. Shandor Monson, 1621 Euclid Ave., Cleveland.
Secretary: Dr. Carl Ellenberger, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Alfred Cowan, 1930 Chestnut St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. H. D. Emswiler, 370 E. Town St., Columbus, Ohio.
Secretary-Treasurer: Dr. D. G. Sanor, 206 E. State St., Columbus, Ohio.
Place: The Neil House. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Arthur Padillo, 414 Medical Professional Bldg., Corpus Christi, Texas.
Secretary: Dr. Edgar G. Mathis, 815 Medical Arts Bldg., Corpus Christi, Texas.
Time: Second Friday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. S. F. Harrington, 921 Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. Abell Hardin, Medical Arts Bldg., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Dr. Howell L. Begle, 2730 E. Jefferson Ave., Detroit.
Secretary: Dr. C. W. Lepard, 1025 David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month, November through April.

Detroit Ophthalmological Society

President: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Place: Club rooms of Wayne County Medical Society. Time: Third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Appointed at each meeting.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Eastern Pennsylvania Association of Eye, Ear, Nose and Throat Physicians

President: Dr. James E. Landis, 232 N. 6th St., Reading.
Secretary-Treasurer pro tem: Dr. Paul C. Craig, 232 N. 5th St., Reading.
Time: Last week in April each year.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Rex Howard, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Gough, Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. Felician J. Slataper, 1110-1111 Medical Arts Bldg., Houston, Texas.
Secretary: Dr. Theo. L. Holland, 611 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. Myron Harding, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Edgar Johnson, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Harold Snow, 614 S. Pacific Ave., San Pedro, Calif.
Secretary-Treasurer: Dr. Oliver R. Nees, 508 Times Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. Sylvester H. Welch, 102 N. Brand Blvd., Glendale, Calif.
Secretary-Treasurer: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Lower Anthracite Eye, Ear, Nose and Throat Society

Chairman: Each member in alphabetical order.
Secretary: Dr. James J. Monohan, 31 S. Jardin St., Shenandoah, Pa.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.
Secretary: Dr. Frazier Williams, 1801 I St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member in alphabetical order.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. Edwin C. Bach, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.
Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. M. M. Cullom, 700 Church St., Nashville, Tenn.
Secretary: Dr. R. E. Sullivan, 432 Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Haven Ophthalmological Society

President: Dr. William H. Ryder, 185 Church St., New Haven, Conn.
Secretary: Dr. Frederick A. Wiess, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Frank C. Keil, 660 Madison Ave., New York.
Secretary: Dr. Willis S. Knighton, 121 E. 61st St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Sigmund Agatston, 875–5th Ave., New York.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., First Monday of each month from October to May, inclusive.

Oklahoma City Academy of Ophthalmology and Otolaryngology

President: Dr. James P. Luton, 117 N. Broadway, Oklahoma City.
Secretary: Dr. Harvey O. Randel, 117 N. Broadway, Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. D. D. Stonecypher, Nebraska City, Neb.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m. dinner; 7 p. m. program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. Thomas Sanfacon, 340 Park Ave., Paterson, N. J.
Secretary-Treasurer: Dr. J. Averbach, 435 Clinton Ave., Clinton, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

President: Dr. Wilfred E. Fry, 1819 Chestnut St., Philadelphia.
Secretary: Dr. Glen Gregory Gibson, 255 S. 17th St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. John B. McMurray, 6 S. Main St., Washington, Pa.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Reading Eye, Ear, Nose and Throat Society

President: Dr. R. M. Brickbauer, Shillington, Pa.
Secretary: Dr. Paul C. Craig, 232 N. 5th St., Reading, Pa.
Place: Wyomissing Club. Time: 6:30 p. m., third Wednesday of each month from October to July.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Peter N. Pastore, Medical College of Virginia, Richmond, Va.
Secretary: Dr. Clifford A. Folkes, Professional Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.

St. Louis Ophthalmic Society

President: Dr. C. C. Beisbarth, 3720 Washington Blvd., St. Louis.
Secretary: Dr. H. R. Hildreth, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting, 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmo-Oto-Laryngological Society

President: Dr. Dan Russell, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. P. G. Bowen, 315 Camden St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Roy H. Parkinson, 870 Market St., San Francisco.
Secretary: Dr. A. G. Rawlins, 384 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Clarence A. Veasey Sr., 421 W. Riverside Ave., Spokane, Wash.
Secretary: Dr. Clarence A. Veasey Jr., 421 W. Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. A. H. Rubenstein, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. H. Blaisdell, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. E. A. Orwig, 420 Madison Ave., Toledo, Ohio.
Secretary: Dr. E. W. Campbell, 316 Michigan St., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. S. Bockoven, 1752 Massachusetts Ave., Washington, D. C.
Secretary-Treasurer: Dr. John Lloyd, 1218–16th St. N. W., Washington, D. C.
Place: Medical Society of District of Columbia Bldg., 1718 M St. N. W., Washington, D. C. Time: 7:30 p. m., first Monday in November, January, March and May.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.

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